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SEVERE UVEITIS WITH ASSOCIATED ALOPECIA, POLIOSIS, VITILIGO AND DEAFNESS

A SECOND REVIEW OF THE PUBLISHED RECORDS

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DETROIT

I wish to express to the officers and fellows of the College of Physicians of Philadelphia my appreciation of the honor they have conferred on me by extending an invitation to deliver the second de Schweinitz lecture. I have chosen to present a second review of the published records of severe cases of uveitis with associated alopecia, poliosis, vitiligo and deafness.

In 1931 I reported 2 cases of uveitis with associated alopecia, poliosis, vitiligo and deafness and reviewed the records of 17 other cases found in the literature, basing my conclusions on the total of 19 cases.[1] Since that time 10 additional cases have been reported. Five of these were reported from Cuba, 2 by Yanes and Ferrer [2] and 3 by Avalos,[3] 1 was reported from China, by Luo,[4] and 1 from South America, by Weskamp,[5] and 3 were reported from the United States, 2 by Rones [6] and 1 by Davies.[7] In addition to these cases, Luo cited 6 other cases reported in Japan, referred to him by Koyanagi, but as I was unable to check on these records the cases are not included in this report. Two of the cases reported by Yanes and Ferrer in 1931 were not included in my original report made that year. By adding the cases reported since 1931 and including the 2 reported by Yanes and Ferrer, there are a total of 29, which number will be considered in the calcu-

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Second de Schweinitz Lecture.

Read before the Section on Ophthalmology of the College of Physicians of Philadelpha on Nov. 16, 1939.

1. Parker, W. R.: Am. J. Ophth. **14**:577, 1931.
2. Yanes, T. R., and Ferrer, O.: Rev. cubana de oto-neuro-oftal. **7**:5, 1938.
3. Avalos, E.: Rev. cubana de oto-neuro-oftal. **1**:242 and 238, 1932; **5**:75, 1936.
4. Luo, T. H.: Chinese M. J. **50**:1409, 1936.
5. Weskamp, R. L.: Rev. Asoc. méd. argent. **46**:1451, 1932.
6. Rones, B.: Uveitis with Dysacousia, Alopecia and Poliosis, Arch. Ophth. **7**:84 (June) 1932.
7. Davies, W. S.: Uveitis with Associated Alopecia and Poliosis, Arch. Ophth. **14**:239 (Aug.) 1935.

lations of the relative frequency of the cardinal symptoms composing the symptom complex of this disease.

The condition in these cases may be defined as a severe uveitis characterized clinically by binocular involvement, a rapid course, a grave prognosis and the association with alopecia, vitiligo, poliosis and partial deafness.

It was my good fortune to observe 1 case of this type of uveitis from ten days after the onset until the eyes were quiet and vision was reduced to perception of light six months later. As the patients in many of the reported cases did not come under observation until rather late in the course of the disease, I shall quote from my original report, giving in detail the clinical aspect of the condition as it developed, and I shall also add an outline of the record of a second case in which the patient did not come under observation until ten months after the onset of the disease.

CASE 1.—A youth aged 18 presented himself at the ophthalmic clinic of the University of Michigan on Dec. 12, 1929. The family and previous histories contained nothing of significance. The patient gave a personal history of having had dimness of vision, photophobia and pain in the eyes for ten days. During this time the vision had rapidly grown worse in spite of treatment. The patient feared that his partial blindness had been caused by the ingestion of about a pint of home-made liquor to which had been added potassium hydroxide.

Vision in each eye was limited to ability to count fingers at 3 feet (90 cm.). Tension was normal, and the pupillary reaction was present but sluggish. Ophthalmoscopic examination of the right eye showed that the pupil dilated evenly, the media were clear and the disk was edematous and swollen about 1 diopter, the rings being obscured mesially. The retina showed loss of transparency, the veins were markedly engorged and the macula was edematous. The pupil of the left eye was only slightly dilated, there were posterior synechiae below and the pupillary remnant skirted the pupillary border down and in. The fundus was similar to that of the right eye. Study of the visual fields revealed moderate concentric contractions of the fields for form, loss of the fields for blue and contraction of the fields for red to within the 20 degree circle and for green to within the 10 degree circle. The blindspot was not enlarged. No scotomas were found.

The patient was admitted to the hospital and atropine instilled in both eyes three times daily. A general examination was ordered. Ten days later the following note was made: There was slight circumcorneal injection of the right eye, the pupil was moderately dilated, the iris was slightly off color and the posterior adhesions were at 4 and 10 o'clock. The left eye showed moderate circumcorneal injection, the pupil was only slightly dilated and the iris was greatly swollen and off color, the exudate being around the lower pupillary border. There were no deposits on the posterior surface of the cornea. The instillation of atropine was increased to every two hours, followed by the use of hot compresses, the intravenous injection of foreign protein (500,000,000 typhoid bacilli) and divided doses of mild mercurous chloride given internally. The following day the pupils were not dilated. A subconjunctival injection of epinephrine hydrochloride was given without effect. Two weeks after the onset of the disease the irides were swollen and nodular. Keratitis punctata was present. Synthetic epinephrine was instilled without effect on the pupils. The injection of foreign protein was repeated.

The urine was normal. The blood showed evidence of slight secondary anemia (hemoglobin content, 75 per cent; red blood count, 4,400,000). The Kahn test of the blood and of the spinal fluid was negative. The basal metabolic rate was —4. Subcutaneous tuberculin test and bacteriologic examination of the blood gave negative results. Oral examination revealed four teeth with an apical pathologic process; the teeth were extracted, but unfortunately no bacteriologic examination was made. Otolaryngologic examination showed a questionable sphenoiditis and septic tonsils. Tonsillectomy was performed eight days after the patient's admission to the hospital. Later, an exploratory sphenoidectomy revealed nothing abnormal. Examination of the genitourinary tract gave negative results. Roentgen examinations of the chest revealed no evidence of parenchymatous tuberculosis. In the right posterior eighth intercostal space there was a distinct area of increased density situated in the middle fold of the lung. Four roentgenograms were made over a period of seven months without demonstrable change in the shadow. The final opinion was that this shadow was due to a localized scar from an old inflammatory process. There was no evidence of active syphilis. Repeated examinations by the internist failed to demonstrate any abnormality.

In spite of the fact that all possible foci of infection were removed and active treatment instituted, the uveitis progressed. Two months after the onset of the disease there was marked exacerbation of all symptoms. Large punctate deposits appeared on the posterior surface of the corneas, the pupillary areas became occluded with exudate and the irides bulged forward. Hypotomy developed. On February 19 (ten weeks after the onset of the disease) it was first noticed that the proximal ends of the eyelashes were gray and an early vitiligo was present on the face, neck and shoulder. During the week a patchy alopecia developed on the vertex. Because this type of alopecia is often associated with syphilis of the central nervous system, a provocative course of arsphenamine was given, but without change in the serologic reaction of the blood or spinal fluid.

Four months after the onset of the disease the eyes were quiet, and the irides were drawn in folds and vascularized. The pupillary areas were filled with exudative membrane. Vision in the right eye was limited to counting of fingers at 2 feet (60 cm.) and in the left eye to perception of moving objects. The lashes and eyebrows were white, and there were several round areas of alopecia. Six months after the onset of the disease no change was observed in the patient's condition.

CASE 2.—A woman aged 25 presented herself at the clinic at the University of Michigan on May 6, 1930, on account of blindness of ten months' duration. In June 1929 she contracted a severe cold, with a sore throat, stiffness of the neck, severe headache and slight pain in the eyes. In two weeks vision had failed so rapidly that she was unable to see to to get around. In July (one month after the onset of the disease) she suffered attacks of vertigo, ringing in the ears and a distinct but not severe deafness, which persisted for about four weeks. About August 1 (two months after the onset of the disease) tonsillectomy was performed. A roentgenogram of the teeth was negative. Three weeks later the antrum on each side was punctured, but no evidence of infection was found. The patient was given a mercurial inunction and potassium iodide. The vision failed slowly but steadily. Three months after the onset of the disease the hair of the head and the lashes and eyebrows began to fall out. The alopecia was largely confined to the vertex, a fringe of hair being left around the nape of the neck and over the ears. The patient lost 10 to 12 pounds (4.5 to 5.4 Kg.) in weight, felt restless and at times experienced an extreme sense of fatigue. In October (six months after the onset of the disease) she began to feel better. The hair began to grow,

coming in almost white. As the original color of her hair was black, the contrast was said to have been most striking. Twelve months after the onset of the disease her friends noticed that her eyebrows were returning to their original color. She had no vitiligo or night blindness.

When the patient was first seen the vision in each eye was limited to perception of moving objects. Tension of the right eye was 44 and of the left eye 48 mm. (McLean). The iris reflex was absent. The right eye showed moderate circumcorneal injection, a clear cornea, a shallow anterior chamber, many deposits on the posterior surface of the cornea, an irregular pupillary bodrer, many posterior synechiae, exudates in the pupillary area, a vascular and swollen iris and a nodule at the base of the iris opposite 10 o'clock. The left eye appeared the same as the right eye except that there was no nodule in the iris. The hair, lashes and brows were gray. Ophthalmoscopic examination showed the fundus reflex present, but no details could be made out.

Examinations, including tests of the blood and spinal fluid, a subcutaneous tuberculin test, urinalysis, bacteriologic examination of the blood, roentgenograms of the chest and teeth, examination for clinical evidence of syphilis and pelvic disorders, gave negative results. Sixteen months after the onset of the disease the patient was still blind. The brows had recovered their normal color, but the lashes and hair of the head, while partially restored, still showed marked grayness.

The patient in the first case had no impairment of hearing, and the patient in the second case showed no pigment changes in the skin; otherwise the cases were identical. Rones examined the second patient and included his observations in his study of 2 other patients. All the tests made by him in this case, including those for tuberculosis and syphilis, gave negative results. Rones reported 2 additional cases. In 1, the patient was a Negro boy aged 10. This patient is the youngest one on record. The syndrome was complete except that there was no loss of hearing. Rone's second patient, a white woman aged 28, presented all the cardinal symptoms, the temporary loss of hearing being present in but one ear. Only 1 of the 3 patients examined by him gave a positive reaction to the uveal pigment test.

DAVIES' CASE.—A man aged 31 had bilateral uveitis with temporary loss of hearing coming on one month after the onset of the disease. At the end of two months the hearing was normal. Six weeks after the onset of the disease alopecia developed in patchy areas with poliosis. Six months later the eyes were quiet. Complete posterior synechiae were present. The vision was limited to perception of light. There was no evidence of vitiligo. Examination, including Kahn and tuberculin tests, gave negative results except for the presence of septic tonsils and evidence of poor oral hygiene.

Dr. William Zentmayer will subsequently report an additional case illustrating this type of unveitis. The patient, a woman aged 30, presented all the cardinal symptoms except vitiligo. Photographs of the fundi, however, showed marked depigmentation of each retina and choroid. Possibly the vitiligo had been present and cleared up, as the patient did not come under observation until late in the course of the disease.

COMMENT

In all the cases reported the similarity of the clinical picture was most striking. The uveitis was bilateral, usually appearing in both eyes at the same time. The ciliary injection was not severe, and as a rule the pain was not intense. Fever was rarely present. Alopecia and poliosis were always present; vitiligo was present in 79 per cent of the cases and deafness in 72 per cent.

The ages of the patients in the reported cases varied from 10 to 46, the average age being 30.3. The final visual results were surprisingly favorable considering the severity of the uveitis and the marked loss of vision at the height of the disease. In all the cases the vision was reduced to counting of fingers or perception of light, and 30 per cent of the patients recovered vision of 6/12 or better. Apparently the final visual results depend on the severity of the attack in the individual case rather than on a varied type of infection.

The treatment of the 2 patients who came under my observation included injection of foreign protein, massive doses of salicylates, antispecific medication and tuberculin, no one of which seemed to have any beneficial effect. Atropine, epinephrine hydrochloride and synthetic epinephrine failed to dilate the pupils. Iridectomy was attempted in 1 case. The iris was adherent to the anterior capsule, and a satisfactory coloboma was not obtained. Luo, observing the similarity of the clinical manifestations of this disease to sympathetic ophthalmia, treated his patient after the method of Dr. Verhoeff,[7a] by injections of diphtheria antitoxin. The following doses were administered: 20,000 units daily for one week and once after the lapse of one week. The result was unsatisfactory. The results of treatment have been unsatisfactory in the experience of all who have had the responsibility of the management of cases in this group.

The resemblance of this disease to sympathetic ophthalmia is marked, as in addition to the uveitis, alopecia, vitiligo, poliosis and deafness do occur rarely in cases of sympathetic ophthalmia. No better description of the clinical appearance of this form of uveitis could be given than to say that it resembles a case of bilateral sympathetic ophthalmia. The varied opinions concerning the relation of this form of uveitis to sympathetic ophthalmia are well shown by the following pathologic reports: Ogawa[8] examined the eyeball of a man aged 48 with so-called idiopathic uveitis, the eye having been removed on account of secondary glaucoma. After making a detailed report on his sections, he concluded that the changes did not agree with those of typical sympathetic ophthalmia. On the other hand, Matsuoka[9] in

7a. Verhoeff, F. H.: Arch. Ophth. **56**:28, 1927.

8. Ogawa, A.: Acta Soc. Ophth. Jap. **38**:38-68, 1934.

9. Matsuoka, H.: Zentralbl. f. d. ges. Ophth. **30**:520, 1933-1934.

1933 examined a piece of iris from a patient with uveitis associated with alopecia, poliosis, vitiligo and deafness and concluded that the picture of uveitis was not only clinically but microscopically the same as that of sympathetic ophthalmia.

As in many clinical conditions the causation of which is obscure, syphilis is suggested as the cause of this disease. While all the associated complications met with in this form of uveitis are occasionally seen in cases of syphilis, the test was positive in this series for only 5 of the 25 patients examined, and the majority of observers are of the opinion that syphilis is not usually the cause in this form of uveitis.

Tuberculosis has been suggested as a possible etiologic factor. The patients in the 5 cases reported in this country were all given a subcutaneous tuberculin test, with negative results. In no case was there any clinical evidence of tuberculosis. Weskamp suggested that a filtrable virus was the probable cause but offered no evidence to support his theory.

In the discussion of the clinical manifestations of this disease the presence or absence of glaucoma or hypotony will not be considered, as they evidently are secondary to the uveitis and have nothing to do with the original symptom complex. The presence or absence of neuroretinitis and detachment of the retina reported in a few cases are also omitted for the reason that the incidence of any change in the fundus could not be definitely determined because examination of the tissues involved could not be made in the majority of the cases.

Of the cardinal symptoms, alopecia and poliosis were present in all the cases reported. Most investigators state that a marked loss of the hair of the head occurs as a rule two or three months after the appearance of the ocular symptoms. In 2 of Koyanagi's [10] cases the loss was noted in the third week. He called attention to the fact that if the loss of the hair was not anticipated the earliest appearance of alopecia might be overlooked, which would account for the variable reports in this regard. On the other hand, the bleaching of the brows and lashes is easily noted as an early change, when, as a matter of fact, it probably appears after the hair of the head begins to fall out. In every instance the color of the first growth of hair was white, but there was a tendency for it to regain its normal color. Seven cases are reported in which the hair regained its normal color in from five to eight months.

In general, alopecia may be due to an infection, a reflex neurosis or an endocrine disturbance. The first two types are characterized by a patchy loss of hair (alopecia areata) and the last type by complete baldness (alopecia totalis). As alopecia areata was present in all the cases reported, it is possible that it was due to infection or reflex neu-

10. Koyanagi, Y.: Klin. Monatsbl. f. Augenh. **82**:194, 1929.

rosis. This possibility may offer a suggestion to be followed in the investigation of this obscure disease.

Depigmentation of the skin, vitiligo, like the other component members of the syndrome under discussion, has been observed in association with several types of ocular disease. The first case described was by Gilbert,[11] who reported a case of bilateral optic neuritis progressing to iridocyclitis in a patient with vitiligo, which developed about the time of the ocular symptoms. In 1 of Komoto's cases (cited by Koyanagi [10]) there was marked depigmentation of the choroid. Similar depigmentation of the choroid and retina was seen by Wexler,[12] who expressed doubt that the uveitis could be responsible for the disappearance of pigment of the choroid and retina and was of the opinion that the inflammatory process is more apt to be incidental to disturbed pigment function in the eye. Koyanagi was inclined to the belief that the condition can be compared to an inflammation arising from a trophic disturbance. Demaria,[13] on the other hand, expressed the opinion that the depigmentation has nothing to do with the uveitis but is a mere coincidence and that tuberculosis or syphilis would be found to be the cause.

Vitiligo occurred in 70 per cent of the reported cases. It was noted simultaneously with or immediately after the uveitis and appeared on various parts of the body, usually the head, shoulders, nape of neck and lids. The causation of vitiligo is unknown, but this frequent association with uveitis seems more than a coincidence.

Of the 29 cases reported, temporary impairment of hearing occurred in 69 per cent. In each case loss of hearing came on at the same time or soon after the appearance of ocular symptoms. In the uncomplicated cases the lesion apparently was in the labyrinth or the central nervous system. In the majority of cases the hearing returned to normal. Various theories have been advanced to explain the loss of hearing. It is interesting to note that all the other changes occurred in pigmented tissues, and it has been suggested that accidental or congenital pigmentation of the basilar membrane in the labyrinth may undergo changes, resulting in partial deafness. Normally, the basilar membrane in man is not pigmented.

There is a difference of opinion in regard to whether the uveal tract is first affected, causing a change in the other pigmented structures from anaphylactic toxins formed as an antibody for the infection, or whether all the cardinal symptoms have a common cause. Koyanagi suggested that as the onset of impairment of hearing in his cases occurred regularly in the early stages of the disease, the uveal pigment could not have obtained its antigenic properties in the manner suggested

11. Gilbert, V.: Klin. Monatsbl. f. Augenh. **48**:24, 1910.
12. Wexler, D.: Arch. Ophth. **57**:393, 1928.
13. Demaria, F. B.: Semana méd. **23**:379, 1916.

by Elschnig.[14] Rones performed a uveal pigment test in 3 cases and obtained a positive reaction in only 1. He expressed the opinion, however, that this would not disprove the theory that the disturbance is an anaphylactic one affecting the pigment tissues, as when the patients were examined the disease had already passed the acute stage and the allergy may have been diminished.

While graying of the lashes and deafness have been reported in cases of sympathetic ophthalmia and syphilis, even less frequently does one see associated alopecia, poliosis or vitiligo in cases of uveitis. Koyanagi, after an admirable review of the work that has been done expressed the belief that he is not far wrong when he concludes that in this form of uveitis the hair and cutaneous complications, and undoubtedly also the deafness, are produced, along with the uveitis, by a common and as yet unknown cause.

The incidence of this type of uveitis is not known. In my experience only the 2 cases reported were recognized as cases of a special type of uveitis. The marked similarity of the symptoms in all the reported cases, however, together with the lack of benefit derived from treatment, suggest the possibility that there does exist in this group of cases a definite clinical entity, independent of syphilis and tuberculosis, the causation of which is unknown and for which no specific treatment is available.

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14. Elschnig, A.: Arch. f. Ophth. **76**:509, 1910.

CLINICAL EVALUATION OF TESTS OF DARK ADAPTATION

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The tests which are commonly employed for estimating visual function are carried out with the light-adapted patient. The acuity of the fovea is determined under optimal conditions of illumination, which means that the test chart is illuminated by at least 20 or more foot candles. Similarly, the isopters of the peripheral field are determined with white or colored test objects which reflect sufficient light to stimulate the peripheral portions of the light-adapted retina. Throughout the field areas of depressed function are detected in the photopic retina. The result of these tests allow us to evaluate the functional capacity of the photopic retina.

The retina behaves differently in the dark-adapted state, however, and there are well known diseased conditions in which it is normal when light adapted but is grossly abnormal in semidarkness. Numerous tests have been devised to measure its function in the dark-adapted state, and the literature is now full of the diagnostic value of these tests for various diseases of the eye itself and of the body in general, particularly as relates to vitamin A deficiency. An excellent review of the various types of apparatus used for determining dark adaptation was published by Sloan.[1]

The careful work of Hecht, more than that of any other, has advanced the knowledge of the physiology of dark adaptation to the point where it is applicable to clinical cases. He was the first to show that the cones as well as the rods have the ability of dark adaptation and that the course of dark adaptation is as important to follow as the determination of the

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Read before the College of Physicians of Philadelphia, Section on Ophthalmology, Dec. 21, 1939.

1. Sloan, L. L.: Instruments and Technics for the Clinical Testing of Light Sense: I. Review of Recent Literature, Arch. Ophth. **21**:913 (June) 1939; II. Control of Fixation in the Dark-Adapted Eye, ibid. **22**:228 (Aug.) 1939; III. Apparatus for Testing Regional Differences in Light Sense, ibid. **22**:233 (Aug.) 1939.

final threshold. He has standardized the variables which must be controlled and has demonstrated that unless such controls are kept, clinical work in dark adaptation may lead to erroneous results. He and Shlaer [2] have devised an instrument which more nearly controls these variables than the others which are available.

Because of the disagreement in the literature concerning dark adaptation in various diseased states, it was thought advisable to examine a number of patients with various ocular diseases and a similar group of patients whose eyes were apparently normal but who were hospitalized for ailments in which a deficiency in vitamin A might be expected. The instrument used for these tests was the adaptometer of Hecht and Shlaer, and all the measurements were taken by one of us (R. McD.).

It must not be forgotten that this is a subjective test, which is much more difficult for the subject than having his field of vision taken and scotomas mapped out. Each test takes approximately forty minutes, which is trying for the subject as well as for the operator. When practiced observers are being tested the results for the most part are constant and can be repeated within a small limit of error, but with the ordinary patient seen in the clinic and the wards of a hospital, considerable allowance must be made, and deviations from the normal can be considered pathologic only if they lie well beyond the range of experimental error and can be repeated. This is particularly true of children, many of whom have deficient thresholds at the first examination. In the results which we are reporting repeated examinations were made on all the patients and only deviations from the normal of one-half a log unit or more were considered to be significant.

SIGNIFICANCE OF DARK ADAPTATION TESTS IN DISEASES OF THE EYE ITSELF

(a) Impaired Transparency of the Ocular Media.—A group of subjects with ocular diseases of widely different causation but having in common one factor, i. e., loss of transparency of the ocular media, were selected for examination. In testing dark adaptation the minimal amount of light which will stimulate a known portion of the retina at various time intervals following light adaptation is determined. The size of the test flash and the duration of its exposure are constant. Its intensity is increased until the threshold is reached. It is obvious that less light will reach the retina if the ocular media are cloudy than if they are transparent. Accordingly the threshold will be higher. The course of

2. Hecht, S., and Shlaer, S.: An Adaptometer for Measuring Human Dark Adaptation, J. Optic. Soc. America **28**:269 (July) 1938.

adaptation will not be interfered with, and the cone-rod transition point will remain the same, but the whole curve of dark adaptation will be elevated. The following 2 cases were selected to illustrate this point:

CASE 1.—George S., aged 40, had a history of failing vision of both eyes over a period of two years, gradual in onset. Recently he complained of bilateral monocular diplopia. His general health was good, and physical examination showed nothing save that the patient was prematurely gray and slightly overweight. Vision in each eye was 6/15 with the best correction. The eyes were found to be normal as far as could be seen save for well advanced sclerosis of both lens nuclei and floating opacities in the vitreous. No discrete lesions could be seen in the retina or the choroid through the hazy media. The visual fields were normal.

In this case it was important to obtain some information whether the retina functioned normally and whether the poor visual acuity was due entirely to the loss of transparency of the ocular media. Dark adaptation tests showed the course to be entirely normal, although all the thresholds were definitely raised. This was

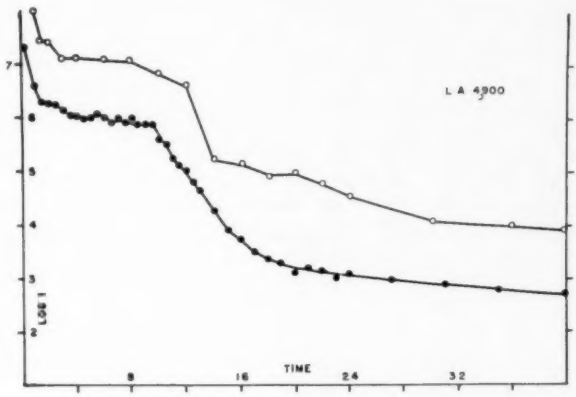

Chart 1.—Light adaptation, 4,900 millilamberts for three minutes. The curve indicated by closed circles represent the course of dark adaptation of a normal person and that indicated by open circles, the course of dark adaptation of George S. (case 1).

undoubtedly due to the lack of transparency of the sclerosed lenses. Had either the cone or the rod portion of the curve been pathologically raised alone, it would have afforded some evidence that the underlying retina was not in good condition (chart 1).

CASE 2.—Miss M. R., aged 30, had increasing corneal dystrophy, similar to the Groenoew type, with gradually failing vision over a period of ten years. For the past year she has been taking large quantities of vitamin A. Vision in the right eye was 6/20 and in the left eye 6/27, each through a pinhole. The eyes were apparently normal save for the opacities in the cornea which occupied the whole cornea with the exception of a clear area around the limbus. The opacities were discrete, round or oval and seemed to consist of infiltrates in and just under the epithelium. Between the denser spots the cornea was diffusely hazy.

The dark adaptation curve was definitely raised throughout the whole course but was otherwise normal. This was probably due to the lack of transparency

of the cornea and is no evidence that the retina itself was not functioning properly (chart 2).

In both these cases, therefore, the raised dark adaptation thresholds must not be considered evidence of either retinal pathologic involvement or of avitaminosis A.

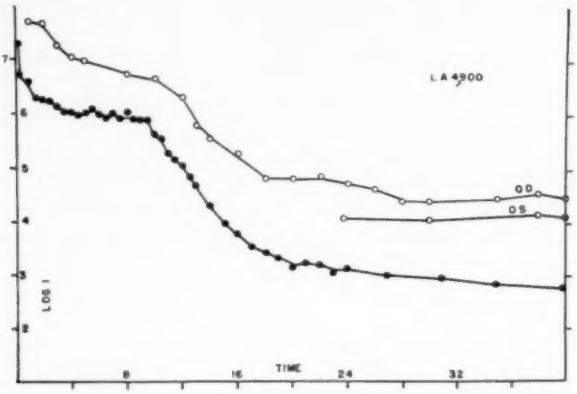

Chart 2.—Light adaptation, 4,900 millilamberts for three minutes. The left eye was not light adapted, but a few threshold readings were taken toward the end of the experiment. The curve indicated by closed circles represent the course of dark adaptation of a normal person and that indicated by open circles, the course of dark adaptation of Miss M. R. (case 2).

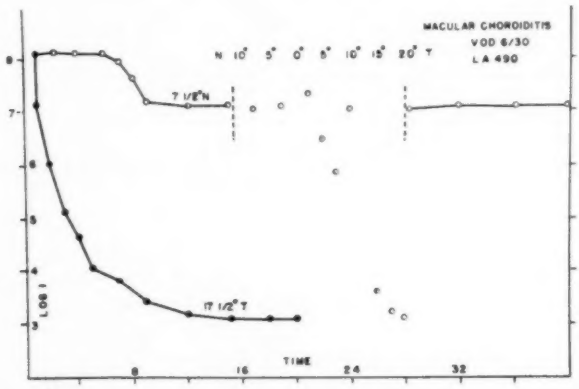

Chart 3.—Light adaptation, 490 millilamberts. At this intensity of light adaptation, cone adaptation is not marked. The curve indicated by open circles shows the course of dark adaptation of S. V. (case 3) measured 7½ degrees to the nasal side of the fovea. The individual open circles inserted in the center represent the threshold readings at various points taken on the nasal and temporal side of the fovea. The curve indicated by the closed circles represents the course of dark adaptation taken with the test flash falling 17½ degrees to the temporal side of the fovea.

(b) Retinal Degenerations.—If the area of the retina selected for testing happens to be diseased, it is reasonable to expect the thresholds found will be abnormal. These thresholds will not represent the thresholds for the rest of the retina, which may be normal. Hence it is important to know that the area of the retina selected for testing in any case is not the seat of some localized inflammation or degeneration. The following cases of localized retinal degeneration will serve as examples:

CASE 3.—S. V., aged 22, had vision of 6/30 in the right eye and 6/20 in the left eye. A diagnosis of bilateral central chorioretinitis was made. Ophthalmoscopic examination showed retinal degeneration about the macula with many fine deposits of pigment. The condition was more advanced in the left eye. The peripheral portion of the retina appeared normal. Physical and laboratory examinations gave negative results, except for a mild frontal sinusitis. Dark adaptation

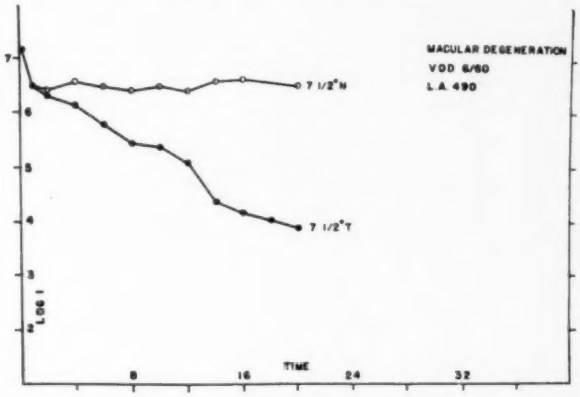

Chart 4.—Light adaptation, 490 millilamberts. Course of dark adaptation of A. G. (case 4) taken 7½ degrees on each side of the fovea.

tested in the usual manner in the right eye 7½ degrees to the nasal side of the fovea showed that there was practically no cone adaptation, followed by poor rod adaptation (chart 3). The final rod threshold was reached at the end of nine minutes and maintained at this level for forty minutes without any further decrease. Following this test, the dark adaptation of various regions of the retina on either side of the fovea was tested. At 15 to 20 degrees from the fovea on the temporal side the dark adaptation thresholds were found to be within normal limits. The subject was then given a second preexposure, and the complete curve of dark adaptation tested for 17½ degrees on the temporal side of the fovea. This resulted in a normal dark adaptation curve.

CASE 4.—A. G., aged 50, had vision of 6/60 in both eyes with correction. He gave a history of failing vision in the left eye for five years and in the right eye for one year. Physical and laboratory studies gave essentially negative results, except for slight roentgen evidence of clouding of the left antrum and infected tonsils. The fundi showed bilateral macular degeneration and pallor of the temporal side of the disk in each eye, which were probably secondary to the macular changes.

Two tests made on the same eye at 7½ degrees to the nasal and 7½ degrees to the temporal side of the fovea showed entirely different curves (chart 4). On the nasal side of the retina there was practically no dark adaptation. On the temporal side adaptation was delayed, but the final threshold reached was fairly low. The visual fields are shown in chart 5.

It should be mentioned that in both these cases central visual acuity was decidedly impaired, which made fixation of the test object difficult. We could not be as positive of the exact location of the retina being tested, therefore, as in normal persons. In spite of this, these cases illustrate very well that one must be sure the area of retina being tested is not diseased before any conclusions can be drawn as to the ability of the eye as a whole to adapt itself to the dark.

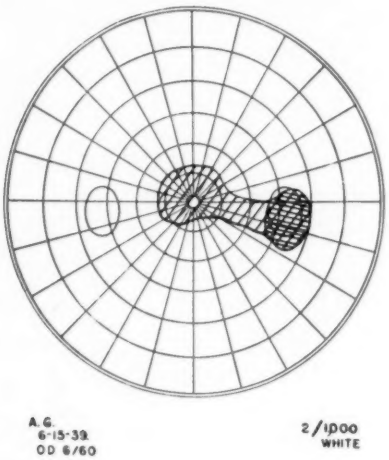

Chart 5.—Central visual field of A. G.

Case 5.—G. B., aged 41, had vision of 6/30 in the right eye and 6/60 in the left eye with correction. A diagnosis of pigmentary degeneration of the retina was made when the patient was 16. Vision has been gradually failing. Ophthalmoscopic examination showed typical changes of retinitis pigmentosa. The central fields were slightly less than 10 degrees with a 2 mm. white test object at 1,000 mm. In the same eye the nasal portion of the retina showed poorer dark adaptation than the temporal retina, though both were considerably abnormal (chart 6).

Case 6.—B. L., aged 24, had vision of 6/6-1 in the right eye and 6/6 in the left eye with correction. A diagnosis of retinitis pigmentosa sine pigmento was made about ten years previously. In 1935 the patient had undergone bilateral superior cervical sympathectomy. The visual fields have continued to contract and are now limited to about 5 degrees with a 2 mm. test object at 1,000 mm. For the past year the patient has received massive doses of vitamin A and carotene, but there has been no regression in the concentric contraction of his fields. Dark adaptation is decidedly impaired, showing some cone adaptation but no rod adaptation at any point within the visual field (chart 7).

CASE 7.—W. J., aged 44, had vision of 6/5 in each eye with correction. The patient complained of eyestrain; on being questioned, he admitted that he sometimes had difficulty seeing in dim light. Ophthalmoscopic examination showed a honeycomb type of depigmentation, like drusen, scattered about the whole fundus and especially marked about the macula. A clinical diagnosis of retinitis punctata albescens was made. The central and peripheral fields were entirely

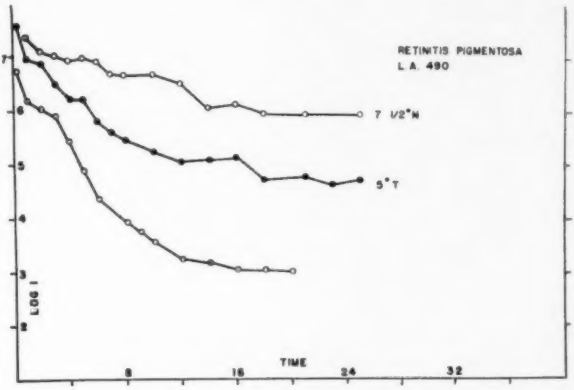

Chart 6.—Light adaptation, 490 millilamberts. The lower curve (open circles) represents the course of adaptation of a normal person. The upper two curves show the course of dark adaptation of G. B. (case 5).

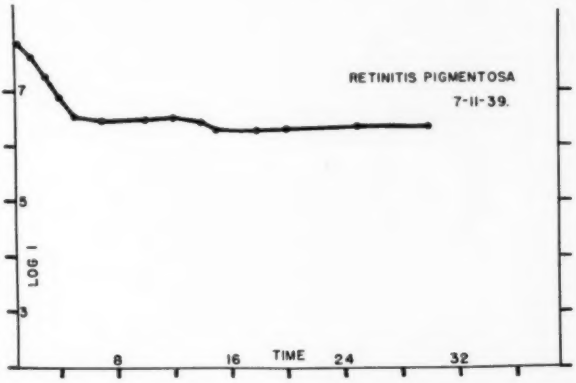

Chart 7.—Light adaptation, 490 millilamberts. The dark adaptation curve of B. L. (case 6) is the same for any seeing portion of the retina.

normal. This is the only patient with this disease we have had the opportunity to examine. There was a marked delay in dark adaptation, but after thirty minutes the subject reached a threshold level ordinarily reached by a normal person in twelve minutes (chart 8). This is in contrast to persons with pigmentary degeneration of the retina whose dark adaptation never approaches normal values.

(c) Refractive Errors.—In examining a fairly large number of normal persons with high refractive errors, a few myopes have been

encountered who on being questioned complained of night blindness and who showed definite raised thresholds. This was particularly true of persons with myopia of 5 to 10 diopters and did not seem to depend on the amount of myopic change seen in the retina and the choroid. High thresholds were found in several cases in which no myopic conus was present and in which no pigmentary disturbance was visible in any portion of the retina.

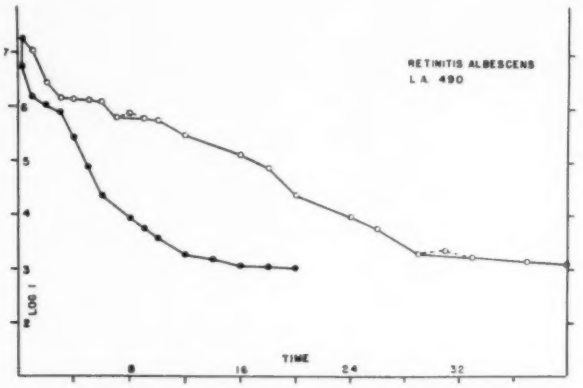

Chart 8.—Light adaptation, 490 millilamberts. The curve indicated by the open circles represents the course of dark adaptation of W. J. (case 7) and that indicated by the closed circles, the course of dark adaptation of a normal person.

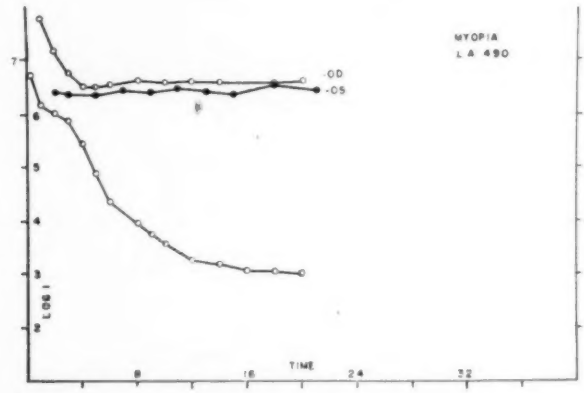

Chart 9.—Light adaptation, 490 millilamberts. The right eye of R. S. (case 8) (uppermost open circles) was light adapted. The left eye (closed circles) was not. The lower curve indicated by open circles shows the course of dark adaptation in a normal person.

Case 8.—R. S., aged 15, had myopia of 12 diopters with no evident pathologic involvement of the fundus except for a moderate temporal conus. Her dark adaptation curve, taken on both the nasal and the temporal portion of her retina, showed only cone adaptation (chart 9), fitting in well with her complaint of

night blindness. The patient was placed on large doses of vitamin A and followed for six months. There was no change in her dark adaptation curve.

(d) Visual Field Defects.—All the patients with glaucoma examined showed no significant change in dark adaptation in the early stages, as long as the central fields were not affected.

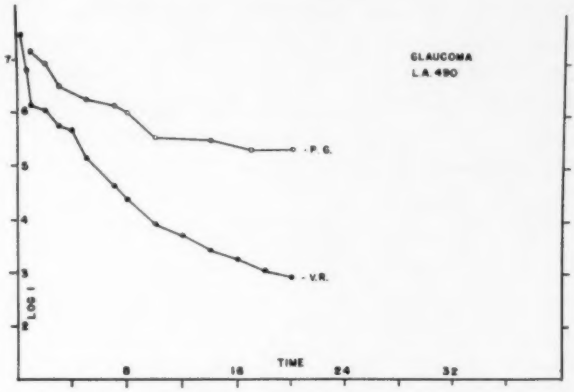

Chart 10.—Light adaptation, 490 millilamberts. The test flash subtending a retinal field of 3 degrees fell on the region 7½ degrees nasal to the fovea in both curves. The curve indicated by the solid circles is that of V. R. (case 9) and that indicated by open circles is that of P. G. (case 10).

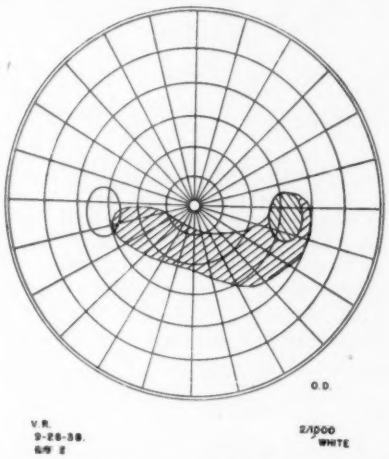

Chart 11.—Central visual field of V. R., which corresponds to the dark adaptation curve (closed circles) in chart 10.

CASE 9.—V. R., aged 15, had vision of 6/6-3 in each eye. This patient has been followed in the clinic for three years and has always had marked cupping of both disks. Tension and visual fields were normal until about one year ago; treatment with miotics was unsuccessful, so bilateral trephine operations were performed. Tension has been maintained within normal limits. Dark adaptation curves have always been normal (charts 10 and 11).

CASE 10.—P. G., aged 56, had vision of 6/9 in the right eye. Vision in the left eye was nil. Ophthalmoscopic examination of the right eye showed a fairly deep glaucomatous cup and moderate angiosclerosis. Tension in the right eye was 31 mm. (Schiötz). An Elliot trephine operation has maintained tension within normal limits. The dark adaptation curve (chart 10) shows a definite rise in the threshold. This is to be expected when one considers the visual fields (chart 12).

No opportunity has been afforded to test various portions of the retina of other patients with Bjerrum scotomas or Rönne steps, but it is likely that one would always find deficient dark adaptation in areas of the affected field and normal dark adaptation in areas which were not yet affected. This is certainly the case in other types of field defects, for example hemianopic fields.

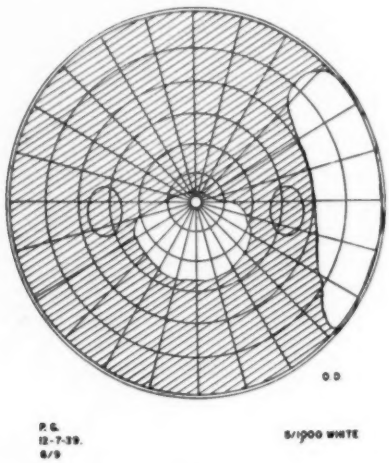

Chart 12.—Central visual field of P. G., which corresponds to the dark adaptation curve (open circles) in chart 10.

CASE 11.—Evans, a patient with bitemporal hemianopia due to a pituitary tumor, had vision of 6/9 in the right eye. Dark adaptation was practically normal in the normal half of the field but markedly deficient near the blind half of the field (chart 13). When the test flash fell onto or near the blind half of the affected field, its intensity had to be raised sufficiently to allow spread of the stimulus into the seeing areas of the retina.

SIGNIFICANCE OF DARK ADAPTATION IN GENERAL DISEASES AND THE INTERPRETATION OF VITAMIN A DEFICIENCY

The role of vitamin A in the formation of visual purple is now well known, and there is ample experimental proof that avitaminosis A leads to night blindness. Hecht,[3] Wald [4] and others have all reported the

3. Hecht, S., and Mandelbaum, J.: The Relation Between Vitamin A and Dark Adaptation, J. A. M. A. **112**:1910 (May 13) 1939.

development of deficient dark adaptation in healthy subjects kept on a diet poor in vitamin A. In our laboratories we have succeeded in producing deficient dark adaptation in a healthy person, who for six months previous to the dietary restriction was examined two to three times a week. He was then placed on a diet containing 300 Sherman units of vitamin A daily. Supplemental vitamins B, C and D were administered. At the end of one week he showed a definite elevation of his cone and rod threshold. This continued for four weeks, at the end of which time there was little further change in this threshold, although the diet was strictly adhered to for another month (chart 14). At the end of two months he was given large quantities of vitamin A by mouth and carotene intramuscularly. No precipitate drop in his thresholds occurred, as has been reported by Wald, but there was a slow return to nearly

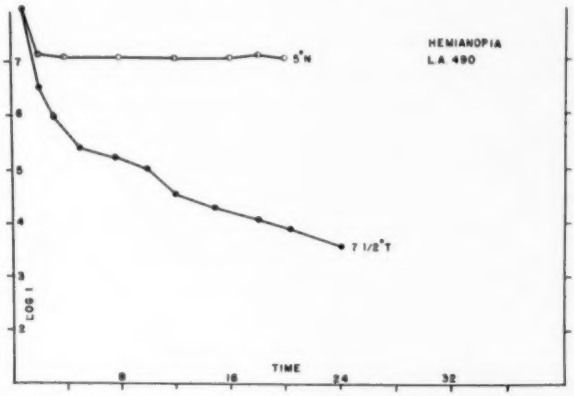

Chart 13.—Light adaptation, 490 millilamberts. The curve indicated by the open circles shows the dark adaptation of Evans (case 11) when the test flash fell in the blind half of the retina, and that indicated by the closed circles the course of dark adaptation when measured in the seeing half of the retina.

normal. The subject has been followed now for nearly nine months since resuming a normal diet, and he has only recently returned to his previous normal threshold. This would suggest the possibility that a marked deprivation of vitamin A may lead to irreversible changes and permanent damage to the scotopic mechanism. Such strenuous attempts to produce night blindness should certainly be approached with caution and a full understanding on the part of the experimental subject of the danger to which he is exposing himself.

4. Wald, G.; Jeghers, H., and Arminio, J.: An Experiment in Human Dietary Night Blindness, Am. J. Physiol. **123**:732 (Sept.) 1938. Wald, G., and Steven, D.: An Experiment in Human Vitamin A Deficiency, Proc. Nat. Acad. Sc. **25**:344 (July) 1939.

Steffens, Bair and Sheard [5] recently maintained 3 healthy subjects on a similar diet low in vitamin for periods up to one hundred and ninety days and were unable to find any significant change in their dark adaptation thresholds, in spite of the fact that sections of skin from one of the subjects showed changes indicative of avitaminosis A. They suggest that normal values of light sensitivity may be maintained over considerable periods of time through the utilization of carotene and vitamin A previously deposited in the skin and fatty tissues. This is somewhat in accord with our finding that some patients whose state of nutrition was so poor that an avitaminosis A might have been expected showed perfectly normal dark adaptation thresholds. A 21 year old girl with anorexia nervosa, who was reduced to skin and bones, her weight being 56 pounds (25 Kg.), had a normal dark adaptation curve.

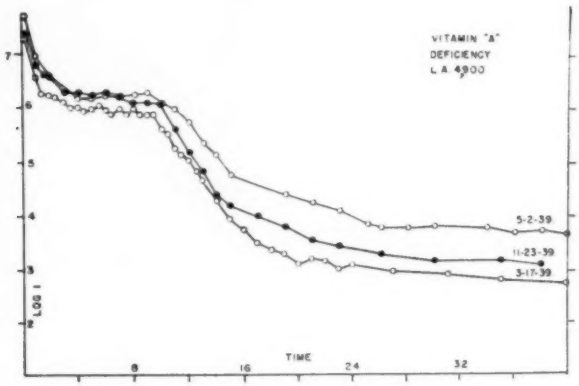

Chart 14.—Light adaptation, 4,900 millilamberts. The lower curve indicated by the open circles shows the course of dark adaptation before a deficient diet was instigated. The upper curve indicated by open circles shows the dark adaptation just before the diet was terminated. The curve indicated by closed circles represents the dark adaptation six months later.

On the other hand, the diagnosis of avitaminosis A is certainly suggested if a person has a deficient dark adaptation curve and no ocular pathologic process is found to explain it. It is probably advisable in the present state of knowledge to make this a suggestive diagnosis. One would expect that a person whose dark adaptation was subnormal due to avitaminosis A would have this promptly restored to normal when given large doses of vitamin A, and yet this is apparently not the case. In the experiment cited previously, the administration of vitamin A did not suddenly restore the subject's light sensitivity, and a number of

5. Steffens, L. F.; Bair, H. L., and Sheard, C.: Photometric Measurements on Visual Adaptation in Normal Adults on Diets Deficient in Vitamin A, Proc. Staff Meet., Mayo Clin. **14**:698 (Nov. 1) 1939.

persons have been carefully followed for whom a presumptive diagnosis of avitaminosis A was made from the dark adaptation tests but who showed no improvement after the administration of large amounts of vitamin A and carotene. It has also been found that it is not possible to improve a subject's dark adaptation when this is already normal by the administration of vitamin A; 1 patient with carotenemia has been examined frequently over a period of one year and has been found to have a threshold no better than the normal.

Twenty patients with a clinical diagnosis of urinary lithiasis were examined, and 3 were found to have abnormal dark adaptation. Two of these 3 patients were operated on; no stone was found in 1 while a calcium magnesium phosphate stone was found in the other, which is the type of stone produced in animals by vitamin A deficiency. Six other patients who were found to have calcium magnesium phosphate stones had dark adaptation curves well within normal limits. Two of the patients in this series who showed poor dark adaptation were given vitamin A concentrate over a period of several months but did not show any appreciable change.

Several cases of hyperthyroidism were followed before and after operation. Two mildly toxic patients were found to have abnormal dark adaptation, while 2 patients with basal metabolic rates of — 80 to — 100 were found to be perfectly normal.

Finally, a group of persons were examined whose eyes were perfectly normal save for low refractive errors, all of whom complained of difficulty in driving a car at night. None of these had any abnormality in their dark adaptation.

CONCLUSIONS

1. Dark adaptation tests are of value only if the condition of the eye being tested is first thoroughly investigated. This should include an examination of the ocular media, the refraction of the eye, a thorough ophthalmoscopic study of the fundus and mapping of the visual fields.

2. If properly controlled dark adaptation tests show a normal course and normal final thresholds, it can be said that the retina behaves normally in the scotopic state. This carries with it the assumption that the vitamin A content of the retina must be normal.

3. If dark adaptation tests show thresholds which are definitely abnormal, the following possibilities must be considered:

(*a*) The subject is a poor observer, either due to inexperience or to lack of intelligence. Repeated tests will usually prove this, as the thresholds in such cases vary markedly from each other at the different examinations. The same or even greater care must be taken in interpreting dark adaptation thresholds as in interpreting visual field defects in poor observers.

(*b*) If the eye is apparently healthy and the subject a good observer, there must be some defect in the retina or its conducting fibers, which present methods of examination fail to reveal, or the retina is deficient in vitamin A.

(*c*) In the present state of knowledge it is entirely proper to say that this suggests a deficiency of the whole body in vitamin A, but until some correlation has been made between the vitamin A content of the blood and the deficient dark adaptation thresholds such conclusions are presumptive and not proved.

DISCUSSION

DR. ROBB MCDONALD: There is one point about the taking of these tests that I should like to mention. In the graphs in which the cone function is well marked and the rod-cone transition time occurred at about ten minutes, the light adaptation was 4,900 millilamberts for three minutes and the test was run for forty minutes. In the shorter graphs in which the cone function is not marked, the light adaptation was 490 millilamberts for three minutes.

There is one other point I should like to mention. Last year, in discussing a paper on dark adaptation here, I stated that in a few cases of suspected vitamin A deficiency I had been able to lower the threshold rather precipitously by the administration of carotene intramuscularly. I have since not been able to repeat this, nor have I been able to reduce the threshold rapidly by the administration of larger amounts of vitamin A. I should like to take this opportunity to correct my former statement.

DR. JACOB B. FELDMAN: I passed the 4,000 mark in dark adaptation studies some time ago. The sharp break in the cone threshold described by Hecht can be likened to 2 persons running at the same speed for several hundred feet. One is a patient with myocardia and the other is normal.

If the pulse of each person is taken every few seconds after the running, the pulse of the patient with myocardia will possibly be 160, 158, 155 and 150 and then will drop to 110 and 108 and continue at 106. The pulse of the normal person will be 120, 118 and 115 and then will drop to 82 and 72 and continue at 72. The running of the two persons may be compared with light adaptation of the two eyes and reading of the pulse, to the dark adaptation. The break in the pulse, 158 to 110 in 1 case and 115 to 82 in the other, is comparable to the final cone threshold of dark adaptation.

Derby and Waite have called attention to this previously. Hecht explained this as a final threshold.

My readings are in millilamberts and the curves plotted are in photons at the suggestion of Dr. Alfred Cowan, who felt that since this is an ocular test the photons give the units of light in units of retinal illumination.

I feel that dark adaptation should be considered more as a test of hepatic function than of a lack of vitamin A.

My experiences have been most peculiar in a case of pregnancy complicated by night blindness. A total of 25,000 units of vitamin A in ten injections cured the night blindness but did not prevent a bad cold from developing.

A total of 1,400,000 units of vitamin A (twenty-eight injections) for cirrhosis did not bring down the pathologic dark adaptation to normal. Of 60 patients with renal calculi, 90 per cent had pathologic dark adaptation. A great many improved clinically, but in not a single case did the renal calculi dissolve.

DR. ALFRED COWAN: I am particularly interested in the findings of Dr. Adler and Dr. McDonald with regard to myopia. It seems unreasonable to consider that in typical myopia the light sense should be reduced. Typical myopia, in my opinion, is nothing but a form of ametropia, and when no other condition is present the dark adaptation should be no higher than in hypermetropia. I should like to know what Dr. Adler thinks of this and whether or not he can offer an explanation; or is it possible that in their cases the myopia might merely have been a coincidence?

DR. FRANCIS ADLER: Deficient dark adaptation might be expected in cases of myopia in which degenerative changes were present in the retina and choroid, but we found a few myopes with definite night blindness in whom no such changes were present. Dr. Hecht has also noted this in a number of myopes he examined. He considered the night blindness in these instances as congenital. I can only say that as yet we do not know how to explain either occurrence.

BOECK'S SARCOID OF THE PALPEBRAL CONJUNCTIVA

MARTHA RUBIN FOLK, M.D.
CHICAGO

Boeck's sarcoid of the palpebral conjunctiva is a rare condition. Boeck [1] first differentiated the disease in 1899 as "multiple benign sarcoid of the skin."

There have been few cases of this type reported in the literature, and when such a case was observed at the University of Illinois I felt that it would be of value to report it.

REPORT OF A CASE

F. M., aged 49, white, married and with five children, all living and well, presented himself at the University of Illinois College of Medicine on Aug. 7, 1939. He complained of a gradual development of redness in his right eye for the past six months. He did not have any pain or discharge.

Twenty years previous to the present complaint his cervical glands became swollen and enlarged gradually. They did not suppurate and caused no pain. After eight months they were removed at the Cook County Hospital, but the hospital was unable to furnish a pathologic report of the condition as the record was lost. On July 9, 1928, he was readmitted to the same hospital, complaining of pressure in the abdomen and vomiting. A diagnosis of peptic ulcer was made. Sippy treatment was instituted, under which he recovered. He was discharged from the hospital on July 13, 1928. Since that time he had had no other symptoms. His family history was unimportant.

Ocular examination revealed vision of 0.5 in the right eye; with correction it was 1.0. Vision was 0.5 in the left eye and with corrcetion 1.2. There was a slight narrowing of the right palpebral fissure. The margins of the lids were red and thickened. The palpebral conjunctiva appeared beefy red and hyperplastic. Rounded outgrowths of granulation tissue were found springing from the palpebral fornices. The bulbar conjunctiva was moderately injected, and symblepharon formed on both sides of the cornea about 3 mm. from the limbus. The cornea was clear, the iris was gray, the pupil reacted to light and accommodation, and the disk and tactile tension were normal. Examination of the fundus did not reveal any pathologic condition.

Conjunctival cultures and smears showed large gram-positive diphtheroids and a few diplococci. Staphylococcus albus which fermented mannite was also found. The diphtheroid was the predominating organism in the smears and cul-

From the Department of Ophthalmology, University of Illinois College of Medicine.

1. Boeck, C.: J. Cutan. & Genito-Urin. Dis. **17**:543, 1899.

tures. Many smears for acid-fast bacilli were negative. A direct smear from the patient's conjunctiva to a rabbit conjunctiva was made; also a pure culture of diphtheroid bacilli was injected subconjunctivally in the left eye. Neither eye showed any inflammatory changes in forty-eight hours.

Physical examination showed a slightly obese man. He had mild hypertension, 180 systolic and 95 diastolic. A roentgenogram of his chest showed no pathologic involvement; enlargement of the left ventricle of the heart was questionable. There were scars at the site of operation on the cervical gland, but no enlargement of any of the glands. He had chronic pyorrhea.

The reaction to the Mantoux test was 2 plus, and the reaction to the Kahn test was negative. Urinalysis gave negative results. The blood picture showed 4,900,000 red blood cells, 9,300 white blood cells, with 20 per cent lymphocytes, 9 per cent monocytes, 47 per cent neutrophils, 7 per cent eosinophils, 1 per cent basophils and 16 per cent stab forms.

Microscopic examination of a biopsy specimen (made by Dr. E. B. Krumbhaar) showed a round section 3 mm. in diameter which stained with hematoxylin and

Fig. 1.—Appearance of the patient.

eosin. Low power magnification showed it to be lymphoid tissue, mostly surrounded by keratinizing squamous epithelium, which sent thin projections deep into the tissue. High power magnification showed the main mass to be made up of several types of cells, of which the commonest had a round, deeply staining nucleus, slightly larger than a small lymphocyte, with a moderate amount of slight, basophilic cytoplasm. A number of larger cells with similar nuclei and cytoplasm were found. They mostly had oval, large nuclei; some of the cells were kidney shaped or multinucleated. None were typical Sternberg-Reed giant cells, though they approached them in appearance. There were also lymphocytes and occasional polymorphonuclear and plasma cells. The delicate fibrous tissue was definitely increased in one area and was scanty in most places. The epithelium was hyperplastic and contained some lymphocytes and polymorphonuclears, and one border was ulcerated.

The dopa reaction was positive.

The diagnosis of Boeck's sarcoid of the palpebral conjunctiva of the right lower lid was made. As Dr. James Ewing stated in a personal communication, "Boeck's sarcoid covers a multitude of miscellaneous lesions." The diagnosis was based mostly on the histologic changes plus clinical evidence and the differential diagnosis.

Fig. 2.—Section from the biopsy specimen; × 100. (This is a different section than the one described in this article.)

In a review of the literature up to 1931 Blegvad [2] could find only 5 cases [3] in which the conjunctiva was involved, and he contributed 3 more. Blegvad stated that Boeck's sarcoid affects mostly the skin, the mucous membrane of the nose, the pharynx and the tonsils and that the condition in 10 per cent of the reported cases was accompanied by iritis or iridocyclitis. Blegvad found his 3 cases of Boeck's sarcoid of the conjunctiva among 20 of the cases of sarcoid seen at the Finsen Institute during the past few years.

In 1910 Darier [4] published an extensive study of sarcoid and divided the disease into four types: (1) multiple benign sarcoid, which may be subdivided into three forms, the large nodular, the small nodular and the diffuse infiltrated form; (2) subcutaneous sarcoid of Darier-Roussy (subcutaneous tuberculid); (3) erythema-induratum-like sarcoid of the limbs (the tuberculin test is sometimes positive in the presence of such a sarcoid), and (4) the Spiegler-Fendt nontuberculous sarcoid with round cells, resembling neoplastic lymphoderma.

ETIOLOGY, PROGNOSIS AND DIFFERENTIAL DIAGNOSIS

Definite etiologic factors are questionable. Several opinions are expressed: 1. The disease may be a peculiar form of tuberculosis, but there is little absolute evidence to substantiate this view. 2. Schaumann [5] upheld the view that the disease is a form of bovine tuberculosis or due to pleomorphic, non-acid-fast tuberculosis organisms. 3. Kissmeyer and Nielsen [6] expressed the belief that the disease is an infectious granuloma approaching more closely to leprosy than tuberculosis in character. 4. Williams and Nickerson [7] suggested that the disease may be due to a filtrable virus.

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2. Blegvad, O.: Acta ophth. **9**:180, 1931; abstracted, Am. J. Ophth. **14**:1280, 1931.

3. Strandberg, J.: Acta dermat.-venereol. **2**:253, 1921. Lehrfeld, L.: Am. J. Ophth. **10**:255, 1927. Schoeppe, H.: Klin. Monatsbl. f. Augenh. **65**:812, 1920. Lutz, W.: Arch. f. Dermat. u. Syph. **126**:947, 1919. Igersheimer, J.: Klin. Monatsbl. f. Augenh. **74**:518, 1925. Schumacher: München. med. Wchnschr. **56**: 2664, 1909. Ehrmann: Zentralbl. f. Haut- u. Geschlechtskr. **9**:167, 1924. Mylius, K.: Ztschr. f. Augenh. **65**:71, 1928; abstracted, Am. J. Ophth. **11**:941, 1928. Habermann: Zentralbl. f. Haut- u. Geschlechtskr. **24**:592, 1927. Hudelo and Rabut: Bull. Soc. franç. de dermat. et syph. **32**:108, 1925.

4. Darier, J.: Monatsh. f. prakt. Dermat. **50**:419, 1910; cited by Derby, G. S., and Verhoeff, F. H.: Arch. Ophth. **46**:312, 1917.

5. Schaumann, J.: Bull. Soc. franç. de dermat. et syph. **41**:1296, 1934; Brit. J. Dermat. **48**:399, 1936.

6. Kissmeyer, A., and Nielsen, J.: Acta dermat.-venereol. **14**:283, 1933.

7. Williams, R. H., and Nickerson, D. A.: Proc. Soc. Exper. Biol. & Med. **33**:403, 1935.

The disease pursues a chronic, sometimes relapsing, course, lasting occasionally for years. Complete recovery may ensue.

In the case reported here, Hodgkin's disease, blastomycosis, tuberculous conjunctivitis and melanoblastoma were excluded by physical examination, histologic, clinical and roentgen studies and the course of the disease.

TREATMENT

In the case reported here large doses of iodides were given for about a month on the supposition that the disease might be blastomycosis. Sulfanilamide was administered for four days without any effect. A series of injections of diphtheroid autogenous vaccine was given. Low voltage irradiation has been used five times up to the writing of this report; the nodular projections of the palpebral conjunctiva are disappearing. Dental treatment has been given for pyorrhea.

Dr. Peter Kronfeld, of Chicago; Dr. E. B. Krumbhaar, pathologist of the University of Pennsylvania School of Medicine, and Dr. James Ewing, director of the Memorial Hospital, New York, studied the biopsy specimens.

MANAGEMENT OF RECURRENT TRACHOMA FOLLOWING SULFANILAMIDE THERAPY

CHEMOTHERAPY COMBINED WITH IONTOPHORESIS THERAPY AS A PROPHYLACTIC MEASURE AGAINST RECURRENCE

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LOS ANGELES

A review of the recent literature with regard to the curative role of sulfanilamide in the treatment of trachoma shows that the data in general rightfully tend to favor the use of this drug. Opinions vary from merely obtaining favorable results to virtually classifying the drug as a specific. Another opinion to be added to the multitudinous clinical reports already published on the subject would be superfluous, but it will suffice to reiterate Lian's [1] summary which appropriately delineates the issue as it appears to be viewed to date: "Although sulfanilamide alone will not cure trachoma, it is a valuable aid in combination with the mechanical methods."

It is the purpose here to deal with an important aspect in the management of the disease during occurrences of late relapses after apparently complete cures with sulfanilamide compounds. Emphasis must be laid on this high rate of relapses, so that clinical opinion as to the reliability of discharging a patient with trachoma as cured will be made with greater reserve. In a limited series of 34 patients, recurrences have been observed in 62 per cent; admittedly this is too small a number to evaluate the probable incidence of recurrence for this particular method of chemotherapy in general, but so striking a ratio from a practical standpoint can hardly be overlooked. Of 21 patients who had recurrences, about half that number had relapses within six months after apparently being completely cured, while the remaining 11 had recurrences in from seven months to two and one-half years. On a basis of these observations, some doubt has to be assumed as to the reliability of certain published statistics on the results of sulfanilamide therapy in cases of trachoma.

In 1935 Seech and I conducted a series of experiments on the action of iontophoresis on rabbits' corneas. After developing a simple and practical apparatus with which to institute this type of treatment, we reported 2 cases of corneal dystrophy in which ionic medication was used with

1. Lian, S. B.: Sulfanilamide in Treatment of Trachoma, Geneesk. tijdschr. v. Nederl.-Indië **78**:1058-1065 (May 3) 1938.

gratifying results.[2] One of us (W. L. C.) continued to carry on further studies with this method of treatment and incidentally used it in 3 cases of dystrophic cornea associated with and resulting from trachoma. While an attempt was made primarily to treat the old corneal ulcers, in 1 case especially in which the trachoma was advanced, not only did rehabilitation of the tissues of the anterior portion of the eyeball begin to occur but definite softening of the somewhat contracted tarsus was also taking place. The thought presented itself to apply this form of therapy in the other 2 cases of trachoma, the trachoma being in a considerably earlier stage. Here an attempt was made both to eradicate the morbific agent and to treat the palpebral conjunctiva as well.

The results were striking. Next the aforementioned series of 34 patients with trachoma were treated and a follow-up study was finally made. Of the 21 in this series who had recurrences, 16 were again treated only by iontophoresis, a 1:200 to 1:100 dilution of quinine bisulfate being used. In the literature reference is made by Selinger[3] to the use of this drug in the treatment of corneal opacities as well as of trachoma, but here it was administered by simple instillation. Edison[4] reported good results in cases of trachoma with zinc ionization; however, in the experiment of Seech and myself[2] repeated use of zinc solution electrolytes not infrequently resulted in considerable untoward tissue reactions.

METHOD OF TREATMENT WITH IONTOPHORESIS

As previously stated, the use of a special but simple storage battery instrument is employed. Its manufacture was described by Seech and myself[2] in a previous publication. More elaborate apparatus suitable for ionization is now on the market if one desires to purchase it. Only the special points of importance will be mentioned here with regard to using this method of treatment for trachoma.

Two drops of a 1 per cent solution of phenacaine hydrochloride or of a 2 per cent solution of butyn are instilled twice at intervals of two minutes in the lower cul-de-sac of the eye. The felt-covered contact area of the positive electrode is dipped into the solution of quinine bisulfate and placed under the lid and up to the fold. Each ounce of the solution, whether the 1 per cent or 0.5 per cent is used, should contain 1 drop of glycerin. It is important that on application of the electrodes they be properly placed so that the maximum benefits will be received

2. Seech, S. G., and Cooper, W. L.: Experimental Iontophoresis of Rabbits' Corneas, Arch. Ophth. **20**:624-640 (Oct.) 1938.

3. Selinger, E., cited, Corneal Opacities, Queries and Minor Notes, J. A. M. A. **107**:375 (Aug. 1) 1936.

4. Edison, S. M.: Trachoma Treated by Zinc Ionization: Preliminary Report, Arch. Phys. Therapy **17**:102-106 (Feb.) 1936.

from each treatment. Throughout treatment the medicated electrode should be in contact with the entire conjunctiva, including the fornices, lids, inner and outer canthi and the caruncle, so that the whole pathologic area is reached during each ionization. The entire area of the lid should be in contact with the moistened electrode.

Electrodes must be well secured to insure proper contact with the patient. The larger, or indifferent, electrode especially should be firmly placed on the upper portion of the back of the neck, if possible, to at least reach the tip of the mastoid bone. This electrode also should be placed on the side opposite to the eye treated; e. g., the right eye requires that the indifferent electrode be placed on the left side of the head and vice versa. Unless the apparatus has a polarity indicator, the polarity should always be tested for, as it is the positive pole which is microbicidal and should be in contact with lesions.

During the half way period of treatment, to keep the positive pole moistened a few drops of the solution of quinine bisulfate should be applied to the pad while it is in place under the lid; this can be done with the aid of a dropper pipet. Likewise, the indifferent negative pole should be kept well moistened with saline solution.

Treatments should be given once every five to eight days; an average of once a week for about ten weeks will usually constitute the complete period of treatment. The exceptions, of course, are those cases in which recurrence has been overlooked for too long a period. As for the amount of current used, it is best to follow the accepted safety rules in the use of the galvanic current generally and allow from 2 to 3 milliamperes per square centimeter of positive electrode surface. The duration of each treatment should be only from two to four minutes, according to the patient's tolerance, an interval timer being used to gage the time. To avoid damage to the tissues, the block tin electrode should be examined before beginning treatments to see that it is completely covered with the felt. Owing to the thin quality of the felt employed, defects in this covering are liable to occur after continued hard usage. In judging the choice of the strength of the solution, the 0.5 per cent solution of quinine bisulfate should be employed for trachoma up to stage II of the MacCallan classification and the 1 per cent solution for trachoma of stage III. By the end of the fourth treatment, judging by its clinical appearance, pannus, keratitis and the arrest of the proliferative process in the granulation tissue will usually be favorably influenced to a marked degree; lacrimation, however, in the majority of cases will continue until the roughened, irritating surface of the affected conjuntiva approximates at least some degree of smoothness. At the end of the sixth treatment complete regression of the follicles can be expected. The average number of treatments for the 16 patients treated for recurrent trachoma was less than 12.

Some patients (5) of this series have remained free from a second recurrence now well over two years since completion of treatment; a second group (9 patients) has had no second recurrence for over one year; the most recently treated group (2 patients) has shown no tendency to recurrence for over ten months. The pertinent question now arises: How long after sulfanilamide chemotherapy should a patient with trachoma wait before being discharged as definitely cured? It must be agreed that the rate of relapse and the time allowed for relapses to occur are still unanswered questions in this early stage of a relatively new type of medication. What is a practical answer to this question? If it is the combination of chemotherapy with an immediate adjuvant treatment in the form of iontophoresis, it should be expected then that permanent results can be obtained. Why wait for recurrence before applying the second or mechanical phase of the treatment? This combined procedure offers, perhaps, the best solution to the problem.

COMBINED CHEMOTHERAPY AND IONTOPHORESIS

The management described here should be regarded as the complete treatment for trachoma rather than merely as a prophylactic measure against recurrence. Little can be added to the suggestions by Harley, Brown and Herrell [5] as far as sulfone therapy is concerned. As a comparatively low concentration of sulfanilamide is required in the blood, between 3 and 5 mg. per hundred cubic centimeters, maintained for about three weeks, dramatic improvement is soon seen. Different persons afford different degrees of concentration with the drug, so that it is well to control the dosage by analysis of the blood. A safe dose rule to follow is ⅓ grain (0.0216) of sulfanilamide for each pound of body weight for the first two weeks and then ¼ grain (0.0162) for the remaining third week. The drug is best administered with grain for grain of sodium bicarbonate. If necessary, the dose can be increased to a daily intake which will maintain a level of approximately 4 mg. per hundred cubic centimeters of blood. Like the observers previously mentioned, I advocate the use of sulfanilamide in preference to any of the other sulfones, but should prolonged medication be required or should the toxic effects of the sulfanilamide result in nausea, headache, diarrhea and vomiting, then the sulfone of choice is azosulfamid (neoprontosil; disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6-disulfonate). By switching to the latter, less dramatic results are obtained, but the lessened toxicity permits continued medication

5. Harley, R. D.; Brown, A. E., and Herrell, W. E.: Sulfanilamide and Neoprontosil in the Treatment of Trachoma, Proc. Staff Meet., Mayo Clin. **14**: 641-645 (Oct. 11) 1939.

with no untoward effects. After the apparent chemotherapeutic cure is obtained, the patient is given at least eight to preferably ten ionization treatments at weekly intervals, as previously described.

SOME IMPORTANT CONSIDERATIONS IN USING THE COMBINED THERAPY

Sulfanilamide therapy cannot be given concurrently with iontophoresis; or iontophoresis should not be begun until the chemotherapeutic phase of the treatment is completed. In experiments with rabbits, regardless of how the dosage of galvanic current was modified or the quinine bisulfate electrolyte diluted, if the animals had any minute concentration of the sulfanilamide in their blood a harsh reaction would result in the ocular tissues after ionization.[6] This reaction is probably due to the presence of the sulfate radical of the electrolyte which combines to form a toxic compound with the sulfanilamide. For this reason a reasonable lapse of time should be allowed after the last dose of sulfanilamide is given, about five days; or if it is desired to start ionic medication sooner, it should be commenced only after the laboratory report for the presence of sulfanilamide in the blood is negative.

During the ionization phase of the treatment greater tissue response to treatment has been found to occur if the patient takes vitamin A in the form of plain or emulsified cod liver oil, 1 ounce (30 cc.) twice daily. If a concentrate must be taken for the sake of convenience, the equivalent in carotene in oil should be prescribed in preference to vitamin A concentrates. In some cases of advanced trachoma in which complete amelioration of symptoms cannot be obtained and the effects of sulfanilamide therapy comes to a standstill, tissue regeneration has been found to proceed more rapidly if the body is fortified with virgin vitamin A or carotene; in these cases iontophoresis should be begun anyway in spite of the arrested and incomplete cure with sulfanilamide. Sometimes pannus will constantly persist, especially in the more chronic forms of trachoma.

Conjunctival instillations of vitamin A is not recommended at any phase of the combined treatment, as photophobia is increased due to the immiscibility of the vitamin oil base with the lacriminal fluid; furthermore, as a result little absorption takes place. Another noteworthy point is that application of sulfanilamide directly to the conjunctival lesions, with or without ionization, affords only mild curative effects, which have not been found lasting.[6]

CONCLUSIONS

In a small series of 34 patients with trachoma treated with sulfanilamide, a recurrence of 62 per cent was encountered within six to thirty months. Sixteen of those with recurrence were subsequently treated

6. Unpublished report.

with iontophoresis, and none had a second recurrence, a majority of the patients having been followed for over two years. The management of these cases is discussed.

It is suggested that routine treatment of trachoma consist of at least eight applications of quinine bisulfate by iontophoresis following apparent cure by sulfanilamide, as a prophylactic measure against recurrence.

The rate of relapse following chemotherapy can be markedly reduced if not completely eliminated by employing ionic medication as a second phase of the sulfanilamide treatment. A few important facts on technic and the adjuvant use of vitamin A are also considered.

ANGIOID STREAKS AND PSEUDOXANTHOMA ELASTICUM

REPORT OF A CASE

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Cases of angioid streaks, and particularly those in which macular changes are present, are sufficiently rare perhaps to justify publication of the report of another case.

REPORT OF CASE

The patient was K. T., a single woman aged 22. Her mother and one sister had poor vision but refused to have their eyes examined. There was nothing relevant in the past history of the patient except that menstrual bleeding had been excessive. She had an alternating divergent squint. Vision was 20/100 in each eye; it improved to 20/30 with a —2.0 sph. —1 cyl., axis 90. There were no central scotomas. The peripheral fields with a 2 mm. test object at 1,000 mm. were typical of those of patients with retinitis pigmentosa. They were tubular in shape, contracted to 15 degrees about the fixation point in the right eye and to 20 degrees about the fixation point in the left eye.

The fundi of the two eyes appeared similar. The disks were normal and were ringed with choroidal pigment. Radiating from the disks were a number of brownish black lines extending toward the periphery; some widened and were irregular in caliber. In places they were about three times the width of a vein. Some were wavy, some branched and still others anastomosed, so that nasally there was an appearance of a network. All of these pigmented streaks were situated beneath the retinal blood vessels, and all seemed to be on the same level, in the deeper layers of the retina. In each fundus the central half surrounding the disk presented a cloudy, grayish appearance, much like the haze seen in retinitis pigmentosa. This partially opaque area was even more accentuated in the neighborhood of the macula. Just temporal to the macula was a profuse conglomeration of small pigment clumps, all in the same layers of the retina. The individual formations were irregular; some were wedge shaped, and others were irregularly circular. In the central part there was a tendency toward coalescence, while in the periphery they remained discrete. A few smaller pigment spots were in the macular region, and a few were to be seen even nasal to the macula. The color of the pigment clumps was muddy brownish black. There were no pigment formations to be seen in the nasal half of the fundus, and the periphery of the retina was normal in appearance. The fovea, however, was free of pigment. There were no hemorrhages in the retina, and the blood vessels were normal.

No opacities were present in the vitreous, and the lenses were clear.

There were also typical lesions of pseudoxanthoma elasticum in the folds of the skin of the neck and axillas.

Presented before the Clinical Society of the Knapp Memorial Eye Hospital, Oct. 29, 1939.

COMMENT

Verhoff expressed the belief that these streaks are ridges, resulting from puckering of the inner surface of the choroid. He considered the puckering to be due to the cicatricial contraction of fibrous tissue which had replaced the deeper layers of the choroid but left the inner layers relatively uninvolved and the fibrosis as probably due to recurrent

Right fundus of patient.

hemorrhages. He suggested the name of fibrosis choroidea corrigans for the conditions.

Hagedoorn,[1] in a recent paper, made the following conclusions: The main pathologic process is a degeneration of the elastic fibers in Bruch's membrane. The degeneration of these fibers impairs the translucency of the membrane. Angioid streaks are ruptures in this degenerated membrane, and the retinal epithelium apparently plays some part in the

1. Hagedoorn, A.: Angioid Streaks. Arch. Ophth. **21**:746 (May); 935 (June) 1939.

clinical appearance of these streaks and pigment clumps. Finally, in cases in which angioid streaks are present a specific degeneration of the elastic tissue system in the body occurs at a time when other cells and tissues are still in good condition. In other words, it is a precocious senility of elastic fibers not only in the eye but, as evidenced by necropsy, in the internal elastic tissue membrane of abdominal blood vessels and in the vessels of the kidney, liver, spleen and other organs.

In these conclusions he is in agreement with other investigators, namely, Böck, Bruch, Grönblad, Marchesani and Wirz.

CALCIFIED CAROTID ARTERY WITH ATROPHY OF THE OPTIC NERVE, CUPPING AND LOW TENSION

REPORT OF A CASE

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In 1932 Knapp [1] reported a number of cases of atrophy of the optic nerve with excavation of the disk and especially with marginal cupping as seen in glaucoma, in which the intraocular tension was persistently low. He was not able to classify them as cases of typical glaucoma and considered "the possible presence of sclerosis of the basal vessels of the brain causing an optic atrophy by pressure." Roentgen examination in these cases showed calcification of the internal carotid, posterior communicating and ophthalmic arteries. The changes in the visual fields had a tendency toward altitudinal defects. The condition was nonprogressive. In a recent publication Knapp [2] reported a number of additional cases and presented the subsequent histories of some of the previously reported cases which were followed up. He came to the following conclusion: "Atheromatous carotid arteries cannot alone cause this descending atrophy, but the condition must be caused by simultaneous circulatory disturbances in the optic nerve from arteriosclerotic vascular changes."

Since these cases of atrophy of the optic nerve with marginal cupping and persistently low tension are not common, I wish to report an additional case from the service of the late Dr. W. W. Weeks, at the New York Eye and Ear Infirmary.

REPORT OF CASE

B. C., a man aged 72, came to the New York Eye and Ear Infirmary on Dec. 5, 1939, to find whether his vision could be improved. In 1934 he had noticed that it was failing, and in 1935 he was told that he had a cataract in each eye. In 1938, after a preliminary iridectomy, the cataract was removed from the left eye. This was followed five weeks later by discission of a secondary membrane. A month later the cataract in the right eye was removed, followed

1. Knapp, A.: Association of Sclerosis of the Cerebral Basal Vessels with Optic Atrophy and Cupping, Arch. Ophth. **8**:637 (Nov.) 1932.

2. Knapp, A.: Course in Certain Cases of Atrophy of the Optic Nerve with Cupping and Low Tension, Arch. Ophth. **23**:41 (Jan.) 1940.

by discission. The patient was given glasses, but his vision remained defective. The intraocular tension in either eye was not elevated during the entire period he was under observation.

On examination it was found that the vision in the right eye was 3/200; with a +7.00 sphere it could be improved to 20/50. In the left eye vision was 6/200; with a +4.50 sphere combined with a +4.00 cylinder, axis 175 it was improved

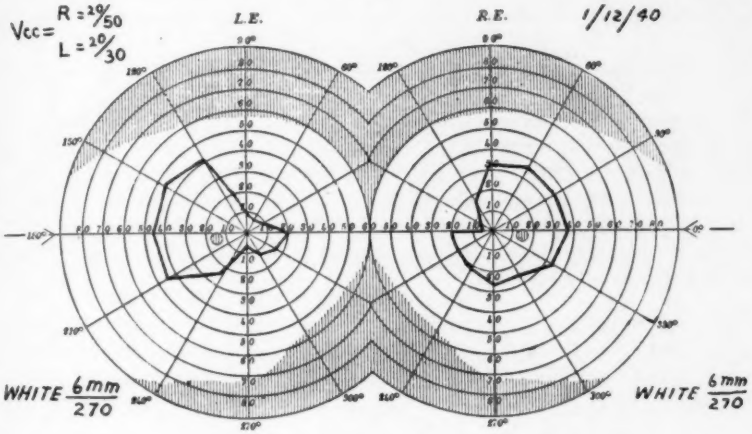

Fig. 1.—Visual fields taken Jan. 12, 1940.

Fig. 2.—Roentgenogram of the skull. The arrows point to the calcified internal carotid artery.

to 20/30. The external ocular movements were normal. The lids, conjunctiva and cornea were normal; the anterior chamber was clear. There were an operative coloboma of the iris, aphakia and a good opening in the secondary membrane in each eye. The vitreous was clear.

Examination of the fundi revealed the following changes: In each eye the disk was pale, with a shallow excavation extending to the margin of the disk,

at the bottom of which the lamina cribrosa was visible. The retinal vessels bent sharply at the margin as they dipped into the disk. There was a halo around the disk. The picture was the one seen in an advanced stage of chronic simple glaucoma. The rest of the fundus was normal except for a moderate degree of choroidal arteriosclerosis.

The intraocular tension measured with the Schiötz tonometer on many occasions was 16 mm. in each eye. I want to emphasize that since the patient had begun to complain of failing vision there had been no increase in intraocular tension.

The visual fields showed a nasal defect and a constricted temporal field in each eye (fig. 1).

The report of the roentgen examination follows: The skull vault was normal in form thickness and structure. The conventional markings were not increased in depth or number. There were deposits of calcium in the carotid artery (fig. 2). No pathologic changes were apparent in the base of the skull, including the sella turcica. The right frontal sinus was well developed. The left was small. Both were clear. The ethmoid sinuses and antrums were clear. The sphenoid sinuses were of moderate size and clear.

COMMENT

The patient presented the ophthalmoscopic and clinical appearance of chronic simple glaucoma: atrophy of the optic nerve, marginal cupping, a halo and bilateral nasal defects with constriction of the temporal fields. The normal intraocular tension and the history of a persistently normal intraocular tension during the entire course of the disease led to the suspicion that this case might be one of atrophy of the optic nerve with calcified carotid arteries. Roentgen examination confirmed the suspected diagnosis. The question whether the atrophy of the optic nerve is due to pressure on the optic nerve by the calcified artery or whether the atrophy is due to a loss of nutrient vessels could not be decided from the clinical examination.

CLINICAL USE OF LACQUER IN OPHTHALMOLOGY

FOR THE TREATMENT OF SQUINT, SUPPRESSION, AMBLYOPIA AND DIPLOPIA

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Lacquer may be applied to an ophthalmic lens in various ways in order to obscure the vision as much as required without an objectionable appearance. This is due to the minute refracting irregularities distributed on the surface of the lacquer as it dries. Lacquer gives the ophthalmologist an additional means of obscuring vision which has some advantages over the standard methods.

The advantages over the use of atropine in the fixing eye are as follows: 1. Distance and near vision are equally affected by the lacquer. 2. There are no allergic reactions. 3. The objectionable appearance of the dilated pupil is avoided. 4. The amount of obscuration of sight can be controlled by the application of the lacquer.

The advantages over a patch are as follows: 1. Binocular vision is stimulated rather than suppressed. 2. The lacquered lens is worn all the time, which is important in the treatment of amblyopia. 3. The field of vision is unimpaired, which today is important as a safety factor for the children who cross the streets.

The disadvantage of a lacquered lens is that a child may do considerable peeking over the top. This often disappears in two weeks if the parents persevere with the child; however, should this fail, celluloid blinders are obtainable which attach around the lens, preventing vision over and around it. The disadvantage of all methods is that it is often necessary to reduce the sight below 20/50, which is not compatible with good school work. For this reason, in these cases it is advisable to start the treatment of amblyopia in June, so that the vacation period will allow enough time for the vision of the poor eye to improve so that school work may be carried on satisfactorily.

The degree of obscuration of sight through the lacquered lens may be varied simply by the amount of brushing or stippling the lacquer receives as it dries. For example, if only a few strokes are made in the lacquer, vision is reduced to about 20/40; however, if a stippling motion of the brush is used as it dries, vision may be reduced as much as 20/100. For patients requiring more obscuration than 20/100, a patch is recommended. An oculist who is interested may perform the following experiment: Several areas of a plane piece of glass are lacquered in

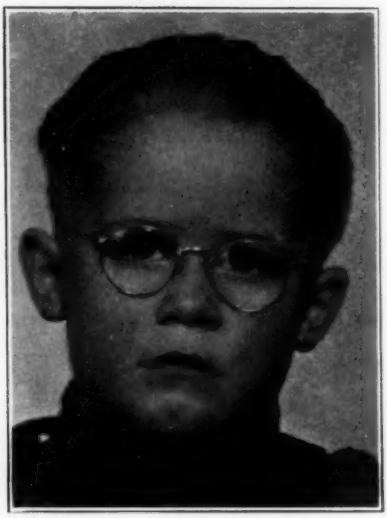

Fig. 1.—Unretouched photograph of a boy wearing stippled lacquer on his right lens, obscuring vision to 20/100. Note that in this view the lacquer is invisible. Reflections of the stippling will be noticed on certain angulations of light just as with any lens under similar circumstances.

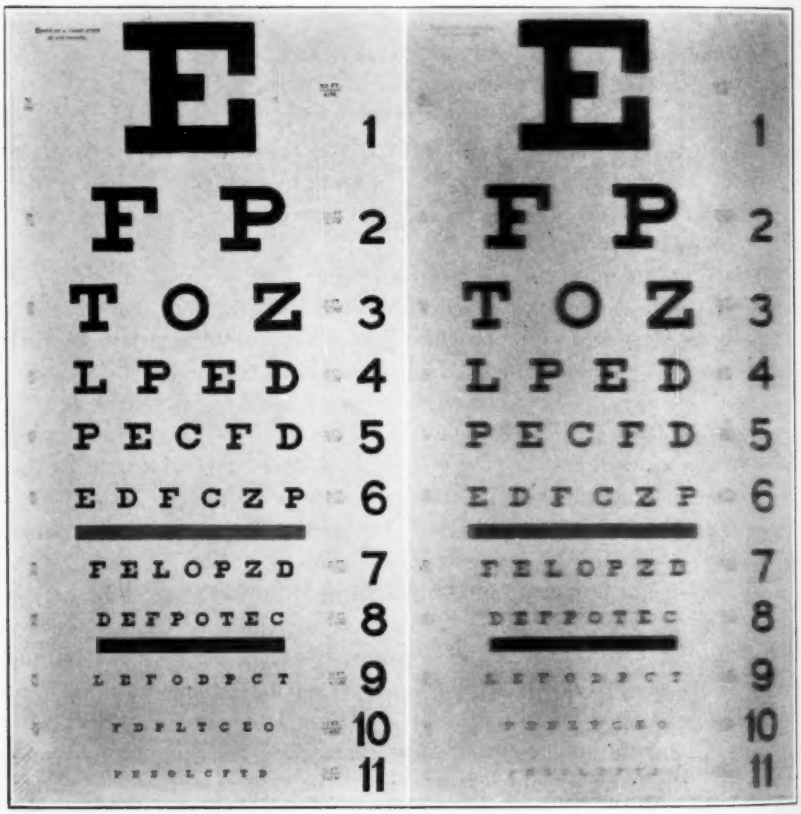

Fig. 2.—A standard test chart is shown on the left. The test chart on the right was photographed at the same distance but with a lacquered plano lens placed before the photographic lens. Note that the blurring is uniform and does not alter the size of the image.

various ways, and the visual acuity through each area is determined. With a little practice the oculist will be able to predict with considerable accuracy the amount of obscuration obtained by the various methods of application.

A stippled, lacquered lense eliminates the disturbances of diplopia. For those patients with diplopia in all directions of gaze, the entire lens before the paralyzed eye must be stippled with lacquer. For those patients with diplopia in one direction only, that portion of the lens covering this area may be lacquered. Patients who have worn frosted lenses appreciate the great improvement in appearance obtained by the lacquered lens.

Lacquer will usually adhere to the lens for about a month, during which time the lens may be washed in cold water. The lacquer may be easily removed when needed by immersing the lens in hot water for several minutes and rubbing with the thumb nail or a similar object.

In my practice lacquer has filled a gap in the treatment of squint, amblyopia and diplopia.

Bottles of lacquer with a brush may be obtained from Belgard Inc., 6 North Michigan Avenue, Chicago.

IRIDOTORSION

CONFIRMATION OF ITS ANATOMIC BASIS BY FORTIN'S INVESTIGATIONS ON GLAUCOMA

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In 1939 Fortin, of Buenos Aires, Argentina, published a monograph on his latest investigations on glaucoma.[1] As only two hundred copies were printed, its interesting contents have remained within the reach of only a few. A good review, however, has been written by Schieck.[2] Fortin had been carrying on his research work for over ten years and was able to present much new material, with many excellent photomicrographs of the ciliary body and its related structures in their relation to glaucoma and its therapy.

According to Fortin, the ciliary muscle is located in a closed cavity, which he calls the iridociliary chamber. The chamber extends anteriorly into the cavities of the iris and posteriorly into the cavities of the ciliary body. It is closed above by the sclera and below by the pigment epithelium. The iris does not contain any stroma between its two layers, but these spaces are simple cavities. As there is no line of demarcation between the iris and the ciliary body, the iridociliary fluid of all the cavities, including the one of the ciliary processes, communicates freely among all of these. It is kept in motion by the ciliary muscle. There is a certain analogy between the ciliary and the heart muscles, the one keeping up the circulation of iridociliary fluid and the other of blood. The ciliary muscle, like the heart muscle, may be subject to occasional attacks of syncope causing transitory glaucoma. The longitudinal fibers of the ciliary muscle are connected with the radiary fibers (sphincter) by intermediary fibers. Both, however, belong together and work in unison. There are no adhesions between the longitudinal fibers and the sclera; they slide along the sclera without any impediment. Those that reach farthest backward (tensor choroideae-Bruecke) are separated from one another by some fluid. They are attached by some fine tendinous strands to the outer walls of the large choroidal veins. They are opened by the contraction of the ciliary muscle and made a "surface of elimination" (*surface d'élimination*-Fortin) for the retina. With the contraction of the ciliary muscle, a slight separation of the radiary and longitudinal fibers

1. Fortin, E. P.: Investigations sur le glaucome, Buenos Aires, El Ateneo, 1939.

2. Schieck, F.: Zentralbl. f. d. ges. Ophth. **42**:380, 1939.

takes place, the radiary fibers moving downward and forward. In this way Fontana's spaces are opened. With the impairment of the ciliary muscle, the functioning of Fontana's spaces stops automatically. The ciliary muscle, stated Fortin, is generally considered the muscle of accommodation. Nature, however, is ingenious and sometimes may use one and the same organ for entirely different ends.

The arteriae ciliares posticae longae, springing from the arteria ophthalmica, should be called arteriae ophthalmicae ciliares, to emphasize their important relation to glaucoma. They and other arterial branches traverse the ciliary muscle and eventually form the circulus arteriosus, while the veins pass only through its neighborhood, never entering the muscle but remaining outside of it. This intramuscular transit of the long ciliary and other arteries has an important bearing on the development of glaucoma; since the contraction of the ciliary muscle also compresses the arteries, it renders them ischemic. At the same time it opens the veins, so that they become distended with blood. Thus the tension is lowered. For the same reason, reading, as a means of inducing contraction of the ciliary muscle, has always been considered therapeutically useful in glaucoma. As already stated, Fortin attributed to the veins of the choroid with their thin walls the role of filtrating areas of the retina. Not only these veins are opened by the contraction of the ciliary muscle (longitudinal fibers) but also the sclerocorneal interstices around Schlemm's canal and the subscleral spaces, via which transudate is being eliminated. The understanding of this mechanism facilitates the understanding of the action of the miotics and the mydriatics. The former contract the ciliary muscle and the arteries, open the veins and lower the tension, while the latter paralyze the muscle, open the arteries, create hyperemia and increase the tension.[3] The ciliary muscle not only moves the iridociliary fluid within the cavities of the ciliary body and iris but also pumps it via the recesses of the ciliary body and iris into the posterior and anterior chambers and into Schlemm's canal. The sphincter pupillae is an associate, although of only secondary importance. The insertion of the ciliary muscle is attached as a fibrous ring to the lower posterior wall of Schlemm's canal. It is only at this place that the ciliary muscle is fixed to the sclera, along which its longitudinal fibers otherwise slide with the contraction of the muscle.

What was formerly believed to be a pectinate ligament is not a ligament but a pectinate filter (*filtre pectiné*-Fortin). It consists of many layers of netlike meshes and contains peculiar cells. Fortin expressed the belief that these may have something to do with the regulation of filtration. By the contraction of the ciliary muscle, the pecten filter becomes dilated and Schlemm's canal is opened. The ciliary muscle,

3. The arterial hyperemia produced by atropine may thus explain its healing effect in cases of iritis.

the pecten filter and Schlemm's canal are therefore important factors in the maintenance of normal tension. As soon as one of them gets out of order, their circle is disturbed and the tension may rise. In which of the closed chambers (*chambres cloisonnées*-Fortin) of the eye the increase in tension will start first cannot be stated; but it is probable that it will be in the ciliary body.

The statements made by Fortin are fully substantiated by beautiful, partly colored photomicrographs. They show the ciliary body and its related structures not only in their normal state but also in a miotic and in a mydriatic state. As regards the practical application of his findings to operative procedures, Fortin contended himself with iridectomy only:

> Iridectomy splits open the iridociliary chamber and facilitates the flow of the fluid from the ciliary processes into Schlemm's canal. Its success depends upon the intact condition of the pecten filter and of the ciliary muscle. The site where it is performed, should be carefully noted in the statistics, as the excision of one or the other of the long posterior ciliary arteries should appreciably lessen the blood supply and may thus prevent an imminent attack of glaucoma.

Donders expressed the belief that glaucoma was due to excessive secretory function. Lagrange mentioned some cases in which the edematous swelling of glaucomatous eyes could fairly be felt by the palpating finger. This is not astonishing when the richness of the ciliary processes in blood vessels is considered. The excess of secretion, according to Donders, would be demonstrated by an irritation of the ciliary processes. Pressed into the iris cavity, the fluid deforms it, the iris moves forward, the pupil becomes dilated and the anterior chamber is obliterated. Disorders of secretion, as induced by an impaired integrity of the walls of the choroidal vessels, swelling of the choroidal blood cushion, sclerosis or obstruction of the scleral interstices, may also affect the regular elimination of the fluid—*"c'est l'étude de leur ensemble qui est celle du glaucome"* (Fortin).

The first paper on iridotorsion was published by me in 1930 [4] and the second in 1937.[5] Like the Lagrange and Elliot operations, iridotorsion consists in cutting a hole in the sclera to connect not so much the anterior chamber as the iridociliary chamber with the subconjunctival space. According to Fortin, there is no line of demarcation between the iris and the ciliary body; all their cavities are connected, and their fluids freely communicate with one another. After the right iris pillar is cut, its double length is used as a channel (with a hoselike arrangement of the two layers of the iris) to convey the iridociliary fluid beneath the conjunctiva. Occlusive constriction of the wick is prevented by a cleancut sclerectomy. Iridotorsion is not an incarceration operation like

4. Denig, R.: Arch. f. Ophth. **125**:156, 1930.
5. Denig, R.: Klin. Monatsbl. f. Augenh. **99**:1, 1937.

Holth's iridencleisis. On the contrary, incarceration has to be prevented by all means, as it would menace the patency of the channel and obstruct the drainage of the iridociliary fluid. In former years I have performed iridencleisis without much satisfaction. I found the operation to be unreliable and dangerous.

Iridotorsion is not a fistulation but a filtration operation. Seidel's fluorescein test revealed the surprising fact that fistulation after the Lagrange and Elliot operations often takes place not under the conjunctiva but straight through it. This explains why one sees a number of cases of late infection. With iridotorsion, the opening in the sclera is closed by an iris cushion. This acts as a wall against infection. It is known that scars filled with uveal pigment do not so readily become infected. Among the patients operated on by me—not quite 200—I have seen those whose eyes were affected with acute purulent conjunctivitis and oozing hordeolas just opposite the cushion, but in none of these did the infection spread to the inside of the eyeball and destroy it. The iris cushion and the instillation of a 20 per cent solution of mild protein silver every four hours saved it from disaster.

Lagrange insisted on a 2 mm. sclerectomy spur for his operation. In cases of high tension such a width must invite prolapse of the ciliary body and vitreous and the dislocation of the lens, which are dangerous complications and spell the ruin of the eye.

Not a few cases of sympathetic ophthalmia have been observed after Elliot's trephining. Up to 1937, 12 cases were recorded in the literature. Not only is there danger of injuring the lens, the ciliary body and the vitreous during the operation, but there is danger of a late infection or chronic cyclitis developing after operation.

With cyclodialysis, the insertion of the ciliary muscle is perforated by the spatula, and the pecten filter is torn off Schlemm's canal. As the regulation of filtration is lost with the destruction of the pecten filter also, the fluid passes, uncontrolled by the pecten filter, with increased velocity into Schlemm's canal. This action explains the subnormal tension that is regularly found after successful cyclodialysis. The accelerated discharge of the fluid should have an effect also on the process of osmosis going on between the vitreous, the lens and the posterior chamber. This will become accelerated, and there will be less time for the exchange of nutritive material. In the course of time nuclear cataract and myopia begin to develop, especially after cyclodialysis has been performed several times. As the process of obliteration of the iris angle goes on, the tension may rise again, sometimes after years of normal values. Cyclodialysis repeated at some other site may lower the tension again if the iris angle is intact. Cyclodialysis is therefore not an ideal operation. However, in cases of a

shallow anterior chamber or stony hard eyeballs, it is an advisable procedure, even if only to lower the tension temporarily and deepen the anterior chamber, to prepare it for iridotorsion.

Figures 30 to 34 in Fortin's monograph are good illustrations of glaucomatous changes in the ciliary body and its related structures. Figure 33, for example, shows "disappearance of the anterior chamber by adhesions between iris and cornea. Schlemm's canal is reduced to a slit. The iridociliary fluid has no outlet into the anterior chamber. The ciliary muscle has formed adhesions which prevent it from functioning; this explains the disappearance of the amplitude of accommodation. The arteriolae are congested."

The indications for iridotorsion are simple and chronic inflammatory glaucoma. It is not indicated in cases of anterior uveitis, hemorrhagic glaucoma and diabetes, nor in cases of atrophic iris or conjunctiva. In cases in which glaucoma occurs after the cataract operation, I always give cyclodialysis the preference. As a rule it proves to be successful, apparently because the absence of the lens results in less crowding of the iris angle, or rather Schlemm's canal. If cyclodialysis fails, I perform iridotorsion. While the usual section for iridotorsion is performed between 10:30 and 1:30 o'clock, in cases of extracapsular cataract extraction it has to be placed far out of reach of possible entanglement with cortical remnants or capsular shreds, i. e., just opposite the section for fear of sympathetic ophthalmia. For the same reason old inflammatory incarcerations and prolapses of iris have to be isolated, as I advised in 1927. Again, Fortin's investigations show the utter futility of such measures as cauterization of the prolapsed tissue.[6]

The object of this paper is expressed by its title. Statistical and other details of iridotorsion are referred to in my paper of 1937. My experiences with iridotorsion during the last three years have continued to be so satisfactory that I do not hesitate to consider it by far the most reliable and the safest operation for glaucoma. The operation is not difficult, even for a novice, if he adheres strictly to the minutest detail of the technic which is presented here.

OPERATIVE TECHNIC

The success of the operation depends entirely on the sclerectomy hole which has to serve as a pipe for the iris wick. The hole does not need to be very wide. In the course of years I found that about 1 mm. width is all that is necessary, if only the entire thickness of the sclera is excised and not merely its superficial layers.

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6. Denig, R.: Klin. Monatsbl. f. Augenh. **79**:599, 1927; Isolation for Iris Prolapse, Arch. Ophth. **8**:477 (Sept.) 1932; Klin. Monatsbl. f. Augenh. **94**: 450, 1936. Markovic, A.: ibid. **98**:187, 1937.

It cannot be emphasized too strongly that this has to be a cleancut (elliptic) hole like the one made in a perfect Elliot operation; otherwise the iris channel will be incarcerated and filtration made doubtful or even impossible. The only way to accomplish this is by measuring the 1 mm. distance from the limbus with a caliper and marking it with a spot of india ink on the sclera. No other way, as for instance, scratching the sclera with a knife, will do, since during the section the scratch will become indistinguishable or nearly so. It must be an easily discernible mark, to act as a "beacon" for the surgeon's eye. With the puncture and contrapuncture at 1 mm. from the limbus, the knife will automatically retain the 1 mm. distance on its way up to the spot of india ink and stay beneath the scleral spur without any temptation to deviate. On reaching the dot, and not before, the knife is turned at an angle of 90 degrees, just outside the black "beacon spot." The various steps of the operation follow:

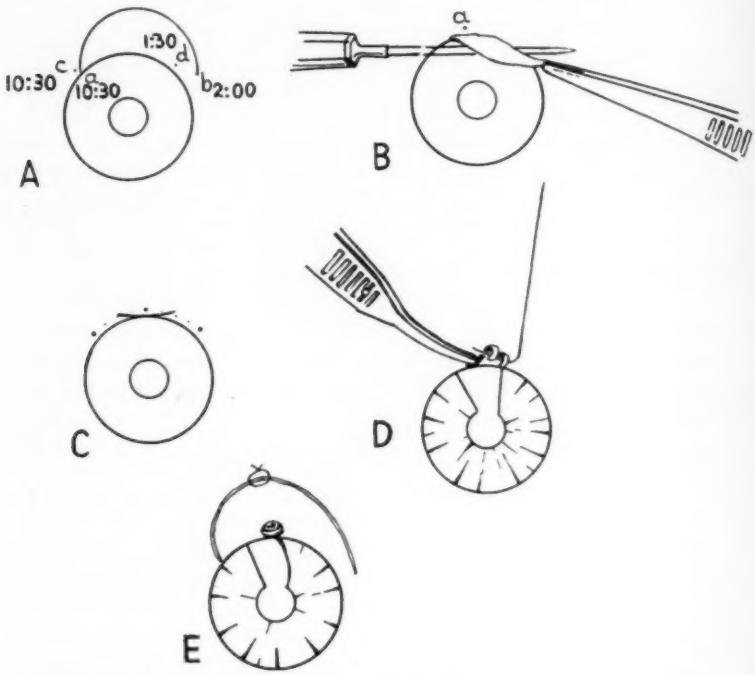
1. The surgeon, standing behind the patient, first prepares the conjunctival flap, as shown in *A*, *a-b* of the accompanying illustration. For the right eye the flap begins at the limbus at 10:30 o'clock, runs up to a height of 8 to 10 mm. and reaches 2 o'clock on the nasal side at a distance of about 3 mm. from the limbus. For the left eye, it begins at 1:30 o'clock at the limbus and reaches 10 o'clock on the nasal side, also about 3 mm. from the limbus. The 3 mm. safety margin on the nasal side insures free communication of the cushion with the remaining conjunctiva. After each step in the operation, bleeding has to be checked most carefully with sponges soaked in solution of epinephrine hydrochloride or, if necessary, by cauterization with the end of a fine strabismus hook heated over an alcohol lamp.

2. With a caliper, the 1 mm. distance between the limbus and the sclera is measured at 12 o'clock. India ink is crushed on a slide. The head of a fine strabismus hook is moistened, and a minute particle of india ink is picked up with it. With the caliper, a slight indentation is made in the sclera, and the particle of india ink is deposited there (*a* in *B* of the illustration). It is then touched with the heated end of the strabismus hook to make it stick to the sclera. The 1 mm. distance is measured once more with the caliper to insure accuracy.

3. The incision should comprise about one fourth of the corneal circumference. It extends for each eye from 10:30 to 1:30 o'clock (*c-d* in *A* of the illustration). A more nasal incision is advisable in cases in which an eventual removal of a cataract may become necessary. The short, sharp Graefe knife is inserted at 10:30 or 1:30 o'clock, respectively, just outside the conjunctival flap and at a point 1 mm. from the limbus. Since with the glaucomatous rigidity of the limbus and the narrowness of the section considerable resistance is offered to the knife, the assistant must exert a gentle pressure on the nasal side of the eyeball with his forceps or make use of a sclerocorneal bridle suture at 3 o'clock for the right eye and at 9 for the left eye. As soon as the surgeon's knife approaches the nasal iris angle, the assistant must take hold of the conjunctival flap and pull it down (*B* of the illustration) in order to clear the way for the contrapuncture, which takes place at a point also 1 mm. from the limbus. It is advisable before making the incision to show the assistant how to handle the flap and the bridle suture. In finishing the section all the surgeon has to do is to look steadily at the spot of india ink and try to reach it with his knife. As soon as he has reached it, he turns the knife at an angle of 90 degrees and brings it out. If the spot should have to be left outside the section because it had by chance been placed beyond the 1 mm. line, it would not matter.

4. While the assistant, to prevent buttonholing, pushes the flap gently down over the cornea with a spatula, the surgeon seizes the sclera at 12 o'clock with a fine straight surgical forceps and excises an elliptic piece with a strong pair of iris scissors (*C* of the illustration).

5. The iris is withdrawn with the blunt iris hook and is immediately seized with the iris forceps held in the left hand and stretched for abscission of the right pillar of the iris. This is done to avoid tearing of the iris by the hook. For the same reason, the iris forceps is not allowed to enter the anterior chamber, since, with the narrowness of the section, injury to the iris and scratching of the capsule of the lens would have to be feared. With the de Wecker scissors (up till then



In *A* the position of the conjunctival flap is indicated by *a-b* and the incision by *c-d*. *B* shows the technic of making the incision. The spot of india ink is indicated by *a*. C shows the location of sclerectomy. *D* illustrates the technic of iridotorsion. *E* shows the iris cushion and the suturing of the flap.

held in the palm of the right hand), the right iris pillar is cut—the temporal pillar of the right and the nasal pillar of the left eye—and carefully placed on the sclera. The right iris angle is pushed back by gentle stroking.

6. Capillary oozing is arrested, if necessary, with the cautery. The pillar of the iris is seized with a fine smooth forceps and, with the pigment layer inside, is wound around a fine dental probe (*D* of the illustration). The iris cushion thus formed is freed from the probe with the aid of the forceps and placed on the sclerectomy hole to close it. If during this maneuver it should have been disarranged, it must be rearranged with the spatula.

7. The flap is secured with one suture only as a rule; otherwise its displacement must be feared (*E* of the illustration). In the taking of this suture, great care must be observed to avoid disarranging the cushion.

Atropine is instilled, and both eyes are bandaged for the first twenty-four hours. The patient may leave the hospital on the fourth or fifth day after the operation.

SUMMARY

The investigations and photomicrographs of Fortin offer an excellent opportunity for analysis of the different operations for glaucoma. They prove unquestionably that iridotorsion is the most logical and anatomically correct procedure.

THE JACKSON CROSSED CYLINDER

A CRITIQUE

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The Jackson crossed cylinder is an outgrowth of an exceedingly clever device called the Stokes lens, which was introduced in 1849.[1] It will be recalled that the first attempt at correcting astigmatism by means of a cylindric lens was made by the astronomer Airy[2] in 1827, only twenty-two years previously. Airy's pioneering step had attracted little of the attention it merited. Cases of astigmatism were still reported as rarities, and cylindric lenses were obtainable from only a few opticians on special order. The pristine trial cases of the day contained few, if any, cylinders and the Stokes lens was invented to fulfil this need. It consisted of two cylinders, one planoconvex and the other planoconcave, placed with the flat surfaces in apposition and so held in a housing that they were capable of being rotated simultaneously in opposite directions. When the axes of the two cylinders were parallel the astigmatic effect was zero, but as their axes diverged from each other a spherocylinder was formed. The astigmatic interval thus created was maximum when the cylinders were at right angles to each other. The net astigmatic effects at various angles as well as the axes could be read off directly from a scale on the margin of the rim. The focal strength of each cylinder was 10 inches, equivalent to about 4 diopters. Thus the original Stokes lens could create spherocylindric gradations between a plano lens and a 4 D. sph. combined with an 8 D. cyl. One determined the axis of an existing astigmatism by rotating the Stokes lens with an arbitrary setting before the eye. Then, keeping the housing fixed for the proper axis, one increased or decreased the strength of the cylinder as required. Allowance had to be made for the spherical by-product.

Dennett[3] in 1885 made a few improvements in the lens mounting and attempted to popularize the Stokes lens as a practical aid in the diagnosis and correction of astigmatism. Hitherto it had "ranked as little better than an interesting toy." This was the situation in 1887, when Jackson[4] realized that the Stokes lens in his trial case could be

1. Stokes, G. G.: Rep. Brit. A. Advancement Sc., September 1849, p. 10.
2. Airy, G. B.: Tr. Cambridge Phil. Soc. **2**:267, 1827.
3. Dennett, W. S.: Tr. Am. Ophth. Soc. **4**:106, 1885.
4. Jackson, E.: Tr. Am. Ophth. Soc. **4**:595, 1887.

used in a manner somewhat different from the original purpose, and from then on a modified form of the Stokes lens has become known as the Jackson crossed cylinder. The scope of its application has been considerably enlarged, and it is the purpose of this communication to examine these uses in detail.

The modern crossed cylinder is a fixed arrangement of plus and minus cylinders at right angles to each other, with a handle placed half way between the two axes. The denominations ordinarily employed are cylinders of 0.12, 0.25, 0.50 and 1.00 diopter. These, respectively, give the following transposed values:

-0.12 sph. $\bigcirc$ $+0.25$ cyl. or $+0.12$ sph. $\bigcirc$ -0.25 cyl.
-0.25 sph. $\bigcirc$ $+0.50$ cyl. or $+0.25$ sph. $\bigcirc$ -0.50 cyl.
-0.50 sph. $\bigcirc$ $+1.00$ cyl. or $+0.50$ sph. $\bigcirc$ -1.00 cyl.
-1.00 sph. $\bigcirc$ $+2.00$ cyl. or $+1.00$ sph. $\bigcirc$ -2.00 cyl.

The two weaker forms are used when the visual acuity is good and the two stronger when it is low. The crossed cylinder is used as an auxiliary lens and may be employed: (1) as a means of building up the optimum spherocylindric combination before the patient's eye, (2) for the determination of cylinder strength, (3) for the determination of cylinder axis and (4) for the estimation of the required reading addition in presbyopia. In all instances in which the crossed cylinder is employed, except in determining presbyopic correction, it is recommended that the patient's vision be fogged with a $+0.25$ or a $+0.50$ D. sphere.

BUILDING UP THE SPHEROCYLINDRIC COMBINATION

Suppose that a spherocylindric combination is placed before the eye and it is desired to test further for possible improvement in the cylindric correction. If a plus cylinder increment should be accepted, the presence of the minus spherical element in the crossed cylinder will enhance the acceptance by reducing the fogging and is an advantage. But if a minus cylinder increment should be accepted, the presence of the plus spherical element in the crossed cylinder increases the fogging effect and may deter the patient from accepting the cylinder. In the latter instance the spherical element is not an advantage.

If a spherocylindric combination is in the trial frame, the crossed cylinder is placed first with the plus axis parallel to the cylinder in the trial frame, and then a quick reversal is made to bring the minus axis in the same line. It is obvious that only two positions relative to the cylinder in the trial frame are thus obtainable, for while the plus cylinder is held in one meridian the minus cylinder lies simultaneously at right angles to it.

For building up the optimum spherocylinder, I prefer to use the individual cylinders taken from the trial case, placing them first parallel

and then at right angles to the cylinders in the trial frame. These maneuvers are done first with a weak plus cylinder and then with a weak minus cylinder. The appropriate changes are made when improvement in vision occurs, and the process is repeated until maximum vision ensues. This is the familiar process of "crossing through" with cylinders and admits of four changes instead of the two which are effected with the Jackson crossed cylinder.

Changes in Trial Frame Setups Produced by the Jackson Crossed Cylinder of + 25 ⊃ — 0.25 D. Compared with Those Produced by Individual Cylinders of + 0.50 and — 0.50 D.

Case	Trial Frame Setup	Effect of Crossed Cylinder	Position of Cylinders	Effect of Individual Cylinders
1	+0.50	A′ +0.25 sph. ⊃ +0.50 cyl., ax. 90	A +0.50 cyl., ax. 90	A″ +0.50 sph. ⊃ +0.50 cyl., ax. 90 *
		B′ +0.25 sph. ⊃ +0.50 cyl., ax. 180	B +0.50 cyl., ax. 180	B″ +0.50 sph. ⊃ +0.50 cyl., ax. 180 *
		C′ +0.25 sph. ⊃ +0.50 cyl., ax. 180 *	C —0.50 cyl., ax. 90	C″ +0.50 cyl., ax. 180
		D′ +0.25 sph. ⊃ +0.50 cyl., ax. 90 *	D —0.50 cyl., ax. 180	D″ +0.50 cyl., ax. 90
2	+0.50 +0.50, ax. 90	A′ +0.25 sph. ⊃ +1.00 cyl., ax. 90	As in case 1	A″ +0.50 sph. ⊃ +1.00 cyl., ax. 90 *
		B′ +0.75 sph.*		B″ +1.00 sph.
		C′ +0.75 sph.		C″ +0.50 sph.*
		D′ +0.25 sph. ⊃ +1.00 cyl., ax. 90 *		D″ +1.00 cyl., ax. 90
3	+0.50, ax. 90	A′ —0.25 sph. ⊃ +1.00 cyl., ax. 90	As in case 1	A″ +1.00 cyl., ax. 90 *
		B′ +0.25 sph.*		B″ +0.50 sph.
		C′ +0.25 sph.		C″ 0*
		D′ —0.25 sph. ⊃ +1.00 cyl., ax. 90 *		D″ —0.50 sph. ⊃ +1.00 cyl., ax. 90
4	+0.50 —1.00, ax. 90	A′ +0.25 sph. ⊃ —0.50 cyl., ax. 90	As in case 1	A″ +0.50 sph. ⊃ —0.50 cyl., ax. 90 *
		B′ +0.75 sph. ⊃ —1.50 cyl., ax. 90 *		B″ +1.00 sph. ⊃ —1.50 cyl., ax. 90
		C′ +0.75 sph. ⊃ —1.50 cyl., ax. 90		C″ +0.50 sph. ⊃ —1.50 cyl., ax. 90 *
		D′ +0.25 sph. ⊃ —0.50 cyl., ax. 90 *		D″ —0.50 cyl., ax. 90

* Positions in which the crossed cylinder and the individual cylinders induce the least changes in the original setup.

The accompanying table indicates the changes in typical trial frame setups produced by the Jackson crossed cylinder of — 0.25 ⊃ + 0.25 D power compared with those effected by the equivalent individual cylindric components taken from the trial case. The differences between them are due to the spherical element in the crossed cylinder.

It is obvious that positions A′ and D′ are in action simultaneously, for while the plus cylinder is at 90 degrees the minus cylinder is at 180 degrees. Positions B′ and C′ are in action simultaneously also, the plus cylinder being at 180 degrees while the minus cylinder is at 90 degrees.

The respective effects of the crossed cylinder and the individual cylinders have been studied to determine which system induces the least changes from the original setup. The one which causes the least abrupt variation from the original sphere and cylinder ought to be the more desirable; slight alterations which might be preferred by the patient could be missed by too great a transition. It is an advantage if the change occurs in either the sphere alone or the cylinder alone or if both are affected for the change to be predominantly in either the sphere or the cylinder. This permits the improvement to be ascribed chiefly to the one or the other. I have placed an asterisk in the crossed cylinder column of the table to indicate the positions in which the crossed cylinder has this advantage, but it will be noted that honors are shared equally with the individual cylinders. It cannot truly be said that the crossed cylinder affords a "purer" test for astigmatism.

While there is a possible saving of time because only two maneuvers are made with the crossed cylinder as against four with the individual cylinders, the former, as has already been noted, yields only two variations as compared to four by the latter. One is not always sure when the patient expresses a desire for a change with the crossed cylinder that the benefit is not derived partly from the spherical element. If the spherical element is to be neglected in determining the direction of change, what is the use of complicating the procedure when simple cylinders are at one's disposal? I do not think that economizing on one or two steps is sufficient reason. If one is to acknowledge the action of the spherical element, the calculation of the new lens becomes very confusing indeed, especially for the beginner.

In using the simple cylinders, when a plus or minus cylinder is preferred, it is often wise before making the change to offer first the cylinder and then a sphere of the same sign and power for comparison. The crossed cylinder does not lend itself to such nice comparisons.

FOR DETERMINATION OF THE STRENGTH OF CYLINDER

The strength of the cylinder is determined by placing first the plus and then the minus elements in the axis of the tentative cylinder. The patient is instructed to concentrate on the smallest line he can read and to report which side of the crossed cylinder blurs the test line the least. The favored side indicates whether the tentative cylinder should be increased or decreased. A slight change is then made, and the process is repeated. When the induced blur is equal in both positions of the crossed cylinder, the corrective cylinder is assumed to be perfect. When the astigmatic error has been fully corrected, the alternate addition of the plus and minus components before the corrective cylinder will cause that meridional focus to fall before and behind the retina by equal amounts. This induces an equal blur in both positions. At the

same time, the other meridian is displaced by the minus and plus elements, but this does not ordinarily interfere with the test.

A discrepancy may occur in a case of myopic astigmatism, axis 180 degrees, when the eye is slightly fogged by 0.25 D. too little minus sphere and when the corrective cylinder in addition needs strengthening by a —0.25 D.

Figure 1 *A* shows the residual undercorrected error without the application of the crossed cylinder. A lens of a —0.25 sph. ⌒ —0.25 cyl., ax. 180 is still required. Figure 1 *B* shows the change made when the crossed cylinder is applied with the minus element at 180 degrees. The undercorrected error is now —0.25 sph. ⌒ —0.25 cyl., ax. 90. Figure 1 *C* shows the change made when the crossed cylinder is applied with

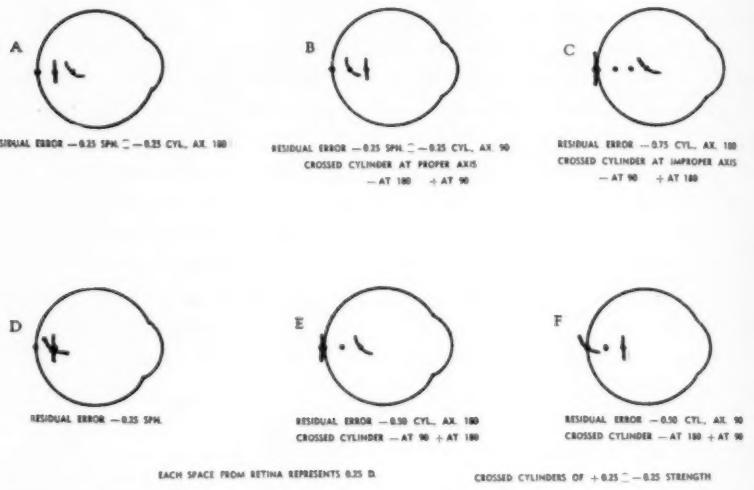
A
RESIDUAL ERROR —0.25 SPH. ⌒ —0.25 CYL., AX. 180
B
RESIDUAL ERROR —0.25 SPH. ⌒ —0.25 CYL., AX. 90
CROSSED CYLINDER AT PROPER AXIS
— AT 180 + AT 90
C
RESIDUAL ERROR —0.75 CYL., AX. 180
CROSSED CYLINDER AT IMPROPER AXIS
— AT 90 + AT 180
D
RESIDUAL ERROR —0.25 SPH.
E
RESIDUAL ERROR —0.50 CYL., AX. 180
CROSSED CYLINDER — AT 90 + AT 180
F
RESIDUAL ERROR —0.50 CYL., AX. 90
CROSSED CYLINDER — AT 180 + AT 90
EACH SPACE FROM RETINA REPRESENTS 0.25 D.
CROSSED CYLINDERS OF +0.25 ⌒ —0.25 STRENGTH

Fig. 1.—Drawing illustrating instances in which the use of the crossed cylinder may be misleading.

the plus element at 180 degrees. The undercorrected error is now —0.75 cyl., ax. 180.

One would expect the patient to prefer the position in figure 1 *B*, since this corresponds with the expected indication for more minus cylinder at 180 degrees. Actually the patient will choose the position in figure 1 *C*, erroneously indicating less minus cylinder at 180 degrees. The explanation is that in this position the vertical meridian is rendered emmetropic. Since test letters are more legible when their vertical components are clear and their horizontal components blurred than vice versa,[5] the position in figure 1 *C* will be accepted. A clear vertical meridian with a badly blurred horizontal axis will be preferred to a

5. Bull, G.: Tr. Ophth. Soc. U. Kingdom **16**:200, 1895-1896.

lesser blurring of both vertical and horizontal axis.[5a] The foregoing conditions may be simulated by placing before the emmetropic eye a lens of + 0.25 sph. ⊃ + 0.25 cyl., ax. 90 and comparing the illegibility of a small test line with the illegibility produced by a + 0.75 cyl., ax. 180.

An inspection of the diagrams will reveal that the same conditions obtain in hyperopic astigmatism, axis 90 degrees, when the astigmatic error is corrected by minus cylinders, at 180 degrees, as is done with the new type of refractometers. Figure 1 *A* would show the sphere fogged by a + 0.50 D. and the cylinder undercorrected by a — 0.25 cyl., ax. 180. In short, in astigmatism with the rule (provided a minus cylinder is used in the correction of hyperopic astigmatism) if the eye is slightly fogged and the cylinder slightly undercorrected, the use of the Jackson crossed cylinder may lead to confusion. Under the same conditions, astigmatism against the rule is not as likely to provoke discrepancies. This is true to a lesser degree also of the individual cylinders. Elimination of the fogging sphere will tend to mitigate the discrepancy, provided the accommodation is inactive. The difficulty in using the Jackson crossed cylinder in an unfogged eye is caused by the stimulus to accommodation exerted by the minus spherical element.

Figure 1 *D* illustrates the condition when no astigmatism exists or when an astigmatism at 90 or 180 degrees has been fully corrected and the eye is slightly fogged. Figure 1 *E* illustrates the axial foci when the crossed cylinder is placed with the minus element at 90 degrees.

The patient will choose the position in figure 1 *E* in preference to that in figure 1 *F*, erroneously indicating more minus cylinder at 90 degrees or more plus cylinder at 180 degrees. Eliminating the spherical fogging will circumvent this pitfall. The effects of the removal of the fogging may be visualized from figure 1 *A* to *F* by displacing the foci 0.25 D. backward.

Crisp,[6] without going into details, has already noted that when the astigmatism lies in the vertical or horizontal axes the crossed cylinder is untrustworthy and has recommended the use of an astigmatic cross instead of test letters. This is a complication in refraction procedure to which objection is justifiable, particularly since the incidence of astigmatism at or near the 90 and 180 degree meridians is frequent.

One must remember that the comparisons of blurring by the patient is a more subjective end point than the optimum visual acuity end point as determined by the ordinary method. The responses with the crossed cylinder are not always consistent with the final prescription. An eye rendered emmetropic will, on being tested with the crossed cylinder,

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5a. Friedman, B.: Acceptance of Weak Cylinders at Paradoxic Axes, Arch. Ophth. **23**:720 (April) 1940.

6. Crisp, W. H.: Tr. Ophth. Soc. U. Kingdom **51**:495, 1931.

prefer the minus to the plus cylinder in the vertical meridian, because by accommodating slightly it will bring the vertical axis into focus. The legibility of the test line will be better than when the eye accommodates for the minus cylinder in the horizontal axis. The eye cannot adjust itself to the superimposed plus cylinder in the vertical meridian. The crux of the question is this: Should the cylinder change as indicated by the Jackson crossed cylinder be prescribed even if it produces no definite increase in visual acuity? It seems to me that the visual acuity should remain the final arbiter in the prescription.

One can simulate the crossed cylinder maneuver by alternately superimposing individual plus and minus cylinders over the trial frame setup. This has the advantage of excluding the extraneous spherical element. The average refractionist can handle his separate cylinders with a rapidity quite comparable to the speed obtained with the crossed cylinder.

DETERMINATION OF CYLINDER AXIS

The Jackson crossed cylinder test employed for determining axis is based on the following principle: When a cylinder is crossed at an angle of 45 degrees by another cylinder of like sign, a new cylinder is formed the power of which is between the separate cylinder powers and the axis of which lies between the axes of the individual cylinders. In addition, a weak sphere is formed. When the Jackson crossed cylinder is used, this weak sphere is practically neutralized by the spherical component of the crossed cylinder and may be disregarded.

Thus, if one crosses two cylinders of + 1.00 D. at 45 degrees, a sphere of 0.3 D. and a new cylinder of 1.4 D., the axis of which is midway between the original axes, are obtained.[7] If the cylinders are of unequal strength, the new axis will veer closer to the axis of the stronger cylinder.

More than fifteen years elapsed after Jackson introduced his crossed cylinder before he appreciated the fact that the lens could be used for determining cylinder axis. He published this discovery in 1907.[8]

In practice, the cross cylinder is so applied before the patient's cylinder that the component of like sign crosses at an angle of 45 degrees. The crossed cylinder is then rapidly twirled to a position 45 degrees on the other side of the cylinder in the trial frame. The patient is asked to report on the clarity of the test line. If one position offers less blur than the other, the cylinder in the trial frame is displaced slightly in the direction of the clearer angle and the process is repeated. When both

7. Thompson, cited by Duke-Elder, S.: The Practice of Refraction, Philadelphia, P. Blakiston's Son & Co., 1935, p. 132.

8. Jackson, E.: Ophth. Rec. **16**:378, 1907.

positions cause equal blurring it is assumed that the axis is correct. The principle of this test is so accurate clinically that I know of no other subjective method of determining axis which is at all comparable.

But since it is desired in this test to increase the corrective cylinder and rotate it to either side of the tentative axis, what could be simpler than to do just that? I temporarily overcorrect the trial frame cylinder by 0.50 to 1.00 D. and move it a few degrees to either side. This procedure is far less complicated and accomplishes optically exactly what the crossed cylinder does. A considerable reduction in time and in the number of maneuvers is the obvious result.

ESTIMATION OF PRESBYOPIC CORRECTION

The crossed cylinder has been utilized as a means of finding the lens which will place the retina in conjugacy with a fixation object at the reading or working distance. The target is a cross formed by multiple vertical and horizontal lines.

Fig. 2.—The Jaques blur point card.

The full distance correction is placed in the trial frame and in front of this a $-0.50 \bigcirc + 0.50$ crossed cylinder is inserted with its respective axes at 90 and 180 degrees. It is recommended that the cylinders take their position against the rule, but I have not found a logical reason for this when the target is a cross and not test letters. For convenience the crossed cylinder is bound in the standard trial lens rim with a short handle. It is assumed that the minus cylinder is set at 90 degrees and the plus cylinder at 180 degrees, and the patient is asked whether the vertical or the horizontal lines of the cross are clearer. If the target distance is conjugate with the retina, the vertical lines will fall as far behind the retina as the horizontal lines fall before it, and the components of the cross will be equally clear.

If the eye is presbyopic, the vertical lines will fall farther behind the retina than the horizontal, and the horizontal arms of the cross will appear blacker. If the patient is overcorrected in his reading addition, the horizontal lines will fall farther in front of the retina than the

vertical, and the vertical arms will appear blacker. The requisite strengthening or weakening of the plus sphere is made to equalize the clarity of the cross arms.

This test has been described by the optometrist Jaques,[9] and subsequent work in this phase of crossed cylinder technic appears to have been done exclusively by optometrists. It has been employed both as a monocular test and as a binocular test. Used alternately before the right and left eyes, it may serve as a check for the distance correction; if unequal additions are required, it is an indication that one of the distance corrections is inaccurate.

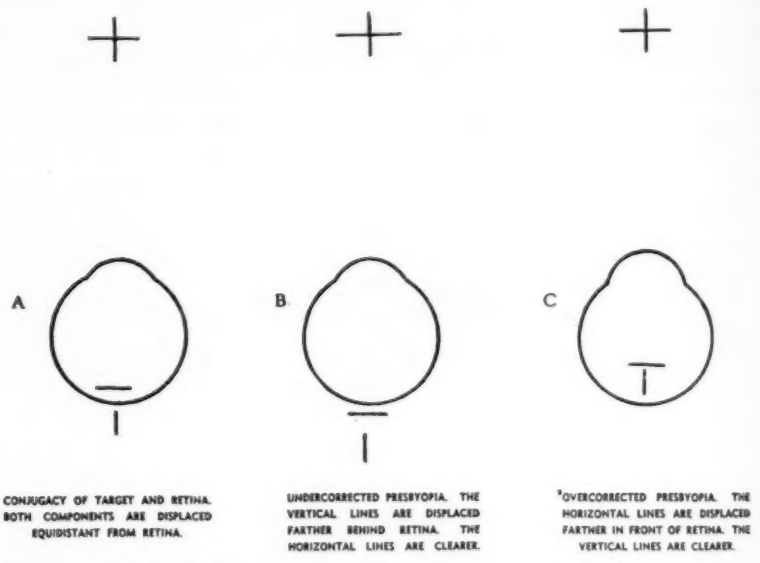
A
B
C
CONJUGACY OF TARGET AND RETINA. BOTH COMPONENTS ARE DISPLACED EQUIDISTANT FROM RETINA.
UNDERCORRECTED PRESBYOPIA. THE VERTICAL LINES ARE DISPLACED FARTHER BEHIND RETINA. THE HORIZONTAL LINES ARE CLEARER.
OVERCORRECTED PRESBYOPIA. THE HORIZONTAL LINES ARE DISPLACED FARTHER IN FRONT OF RETINA. THE VERTICAL LINES ARE CLEARER.

Fig. 3.—Drawing illustrating the use of the crossed cylinder in presbyopia.

My own experiences with this test are not such as to recommend it without considerable reservation. In many instances it checks with the final prescription, but in many cases it indicates too high a spherical addition. This is apparently an expression of the negative accommodation. In still other cases too little addition might be prescribed on the basis of this test. Variable results are often obtained for the same person. It is difficult to obtain an exactly uniform clarity of all the lines, and many patients find it hard to answer with certainty. The astigmatic correction in the trial frame must be meticulously exact in order not to modify the effects of the superimposed crossed cylinders.

9. Jaques, L.: Am. J. Optom. **8**:251, 1931. This test seems to have been a rediscovery on the part of Jaques. I found a description of it in "An Optical Primer," by Charles H. Pixley (Chicago, F. A. Hardy & Co., 1916, p. 249). No statement as to the originator of the method is made.

SUMMARY

In the process of building up the optimum spherocylindric combination for the patient, the use of the individual cylinders from the trial case is on the whole more advantageous than the use of the crossed cylinder.

In determining cylinder strength the criterion of improved visual acuity seems more reliable than the comparative blurs involved in the crossed cylinder test. One must bear in mind that a given dioptric distortion of the vertical and horizontal meridians does not produce respectively equal blurs in a test line. This makes it inadvisable in general to depend on the crossed cylinder test in astigmatism in the vertical or horizontal axis, and in particular in astigmatism with the rule when minus cylinders are employed and when the eye is fogged. If the eye is unfogged, the Jackson crossed cylinder excites the accommodation.

A limited experience with the crossed cylinder technic for determining presbyopic correction does not reveal too encouraging results. Final judgment should be deferred until further data from ophthalmologists are available.

The greatest value of the crossed cylinder lies in the determination of the astigmatic axis.

A simplified test for axis based on the optical principles of the crossed cylinder is offered.

CENTRAL SCOTOMAS

THEIR IMPORTANCE IN TOPICAL DIAGNOSIS

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This paper has been written in an effort to indicate the significance of central scotomas, particularly from the standpoint of topical diagnosis. The study is principally based on clinical cases illustrating the occurrence of central defects in visual fields occasioned by lesions at various levels in the optic pathways.

ANATOMY

While no effort is made to describe the minute anatomic structure of the macula and the papillomacular bundle, attention is drawn to certain anatomic arrangements which account for definite characteristics in cases of central field defects.

Macula.—With the red-free light the macular fibers are seen to follow an arching course above and below the fovea centralis, which they do not cross, to reach the temporal side of the optic nerve, where they form the papillomacular bundle. Localized lesions in the macular region which are readily visible on ophthalmoscopic examination, and with which for the most part we are not here concerned, ordinarily account for pericentral or paracentral scotomas. Either interruption of these macular fibers or vascular obstructions involving the macular area are of particular interest in that each accounts for a characteristic defect in the central field. The blood supply to the macula is, therefore, of interest. The retinal supply is through the upper and lower temporal arteries and in some instances through cilioretinal arteries. At the level of the nerve head crossed and uncrossed fibers are not separated from each other.

Optic Nerve.—The papillomacular bundle, which lies temporally in the nerve head, in the optic nerve contains foveal fibers in its central part with upper macular fibers lying above and lower fibers below. The bundle inclines centrally as it passes posteriorly in the nerve, so that

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when it has reached the level of entrance of the central retinal artery it has attained a central position in the nerve. Here the crossed macular fibers lie to the inner side of the uncrossed fibers. The papillomacular bundle continues to occupy this central position throughout the remainder of the nerve. Just before the chiasm is reached, the crossed macular fibers separate from the uncrossed fibers.[1]

Chiasm.—Since the macular fibers in the chiasm are arranged as a "little chiasma within the chiasma and toward its posterior part," [2] a brief general description of the course of the peripheral fibers through the chiasm may be helpful. In several textbooks, descriptions seem contradictory, in part at least, and in some diagrams are difficult to follow. In order to outline the more important facts regarding the chiasm, we have taken Brouwer's [3] descriptions of his experimental work on monkeys as a principal basis. Crossed and uncrossed fibers separate at the termination of the optic nerve—the anterior angle of the chiasm. The crossed fibers lie medial to the pial septum, which enters the dorsal surface of the nerve at its termination; and the uncrossed fibers lie lateral to this septum. Ventral fibers which cross anteriorly in the chiasm remain ventral and loop into the terminal portion of the opposite nerve. Dorsal fibers, on the other hand, cross farther posteriorly and remain dorsal in position. Before crossing, they form a loop on the homolateral side of the chiasm; this loop is shallower than the ventral loop already described. Uncrossed fibers, both dorsal anl ventral, maintain their relative position in the outer side of the chiasm.[4] Wilbrand expressed the belief that individual fibers from the crossed bundles become associated with individual fibers from the uncrossed bundles after they have passed through the chiasm. He stated that he does not believe that they form a single unit by joining together.

Generalizing, one might say that throughout the chiasm dorsal and ventral fibers, whether crossed or uncrossed, maintain these positions.

Just as is the case with the peripheral fibers, the exact position of the macular fibers in the chiasm is not known with complete certainty. Uncrossed macular fibers occupy a central position in the uncrossed group, and crossed macular fibers occupy a similar position in the crossed

1. Rønne, H.: Pathologisch-anatomische Untersuchungen über alkoholische Intoxikationsamblyopie, Arch. f. Ophth. **77**:1-95, 1910.

2. Traquair, H. M.: An Introduction to Clinical Perimetry, St. Louis, C. V. Mosby Company, 1927, p. 16.

3. Brouwer, B., and Zeeman, W. P. C.: (*a*) Experimental Anatomical Investigations Concerning the Projection of the Retina on the Primary Optic Centers in Apes, J. Neurol. & Psychopath. **6**:1-10, 1925; (*b*) The Projection of the Retina in the Primary Optic Neuron in Monkeys, Brain **49**:1-35, 1926.

4. Wilbrand, H.: Schema des Verlaufs der Sehnervenfasern durch das Chiasma, Ztschr. f. Augenh. **59**:135-144, 1926.

group. The macular fibers cross in the central portion of the chiasm and toward its posterior part. Thus one finds both crossed peripheral and crossed macular fibers occupying posterior parts of the chiasm.

Optic Tract.—In the optic tract the macular fibers, at first lying centrally with the dorsal peripheral fibers above them and the ventral peripheral fibers below them, gradually incline dorsolaterally. They attain this position in the tract just before entering the geniculate body.

Lateral Geniculate Body.—In the geniculate body the macular fibers occupy the dorsal aspect of the body with upper macular fibers lying medial to lower macular fibers. The upper peripheral fibers have come to lie medially and ventrally, and the ventral fibers, ventrally and laterally.

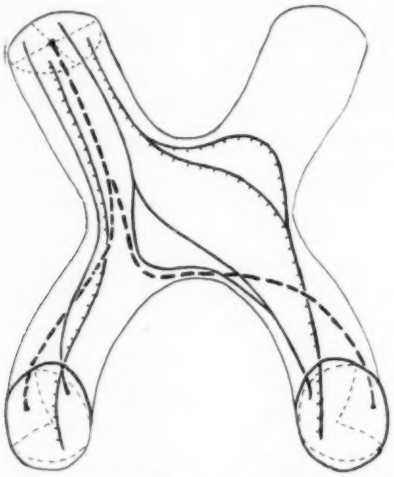

Fig. 1.—Diagrammatic representation of arrangement of fibers in the chiasm. Dorsal fibers are represented by solid lines, ventral fibers by crossed lines and macular fibers by interrupted lines.

Optic Radiation.—Poljak [5] has shown that an intermediate bundle extends from the dorsal portion of the geniculate body to the posterior portion of the calcarine fissure and the posterior pole. The foveal fibers, remaining central, are projected to the posterior extremity.

Where the crossed and uncrossed fibers from the homolateral halves of the retina become associated is not known with certainty. Clinical evidence suggests that association of homologous fibers commences in the tracts (Rønne) and becomes more complete in the geniculate body (Henschen). Incongruity of field defects in the presence of lesions of the temporal lobe suggests dissociation of homologous fibers at this level,

5. Poljak, S.: Projection of the Retina upon the Cerebral Cortex Based upon Experiments with Monkeys, A. Research Nerv. & Ment. Dis., Proc. (1932) **13**: 535-557, 1934.

but congruity of field defects resulting from lesions of the parietal and occipital lobes suggest that they have again become associated.[6] An exception to this generalization is seen in fibers from the extreme nasal periphery which have no corresponding temporal retinal fibers. Brouwer has described the position of these fibers in the geniculate body, and Bender and Strauss [7] have described unilateral temporal field defects as a result of lesions of the optic radiation affecting these crossed nasal fibers. In contrast with these unilateral field defects, central field defects, when due to lesions above the chiasm, are always bilateral.

CENTRAL SCOTOMAS (ANATOMIC CONSIDERATIONS)

In this study we are not considering macular lesions which are readily visible on ophthalmoscopic examination but are almost entirely concerned with what Evans [8] has termed "neurogenic" defects.

In the Macula and Proximal Portion of the Optic Nerve.—1. An involvement of all the structures of the cecocentral area obviously results in a scotoma which is joined to the blindspot, a cecocentral scotoma. Such a scotoma, however, has no localizing value other than to place the lesion below the level at which the crossed and uncrossed fibers have become separated from each other. A cecocentral scotoma in association with contraction of the peripheral field may arise from involvement close to the globe.

2. Interruption of nerve fibers commonly accounts for arcuate defects in the visual fields; such defects are commonly seen in cases of glaucoma. In exceptional cases similar defects are seen to involve macular fibers; and in these instances in an early stage there is a central scotoma bounded by a horizontal meridian which, however, deviates to "spare the macula." A sharply defined arcuate defect is usually properly considered to indicate a lesion at or near the nerve head. Arcuate defects can occur through involvement in the posterior portion of the nerve, where, however, they are not as likely to be sharply defined. An arcuate defect occasioned by a chiasmal lesion has hemianopic characters.

3. A similar scotoma may be observed as a result of obstruction of either the upper or the lower vascular supply to the macula. In such an instance, however, the horizontal meridian of the field "splits the macula." This difference between a vascular lesion and an interruption

6. Harrington, D. O.: Localizing Value of Incongruity in Defects of the Visual Fields, Arch. Ophth. **21**:453-464 (March) 1939.

7. Bender, M. B., and Strauss, I.: Defects in Visual Fields of One Eye Only in Patients with a Lesion of One Optic Radiation, Arch. Ophth. **17**:765-787 (May) 1937.

8. Evans, J. N.: Classic Characteristics of Defects of the Visual Field, Arch. Ophth. **22**:410-431 (Sept.) 1939.

to nerve fibers as observed in central field defects is the single instance where there appears to be a characteristic difference between a vascular defect and a defect due to interruption of nerve fibers.[9]

4. A roughly circular pericentral scotoma is observed as an indication of macular involvement or incomplete involvement of the papillomacular bundle at any level below the separation of crossed and uncrossed fibers in the chiasm. Such a scotoma, when unilateral, always indicates involvement of the optic nerve or macula; if bilateral, the central field defects may be due to bilateral posterior lesions when theoretically the field defects should show hemianopic characteristics.

In the Termination of the Optic Nerve.—A central field defect from a lesion at this level has a hemianopic character through involvement of crossed fibers. Such field defects have been aptly described by Traquair

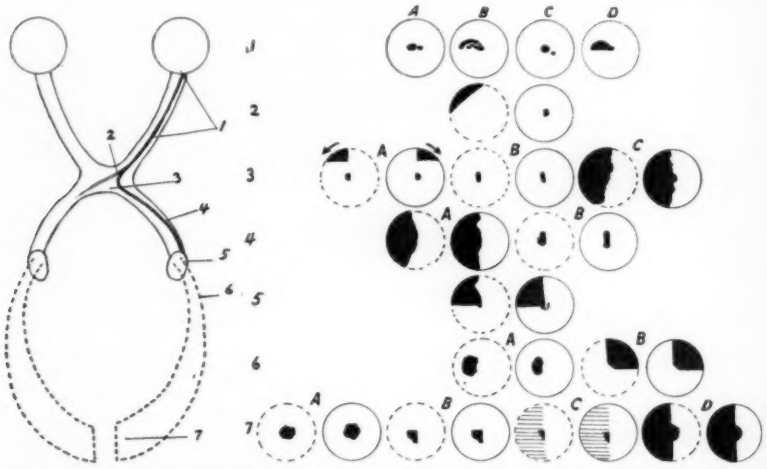

Fig. 2.—Diagram illustrating topical diagnosis of central scotoma, the numerals indicating the area of the optic tract involved and the letters the type of scotoma produced: *1 A*, cecocentral scotoma; *1 B*, arcuate defects; *1 C*, pericentral scotoma; *1 D*, central scotoma due to vascular obstruction; *2*, central defect in the anterior angle of the chiasm with a defect in the field of the opposite eye due to involvement of ventral crossed fibers; *3 A*, bitemporal central and peripheral defects due to involvement of the posterior part of the chiasm; *3 B*, homonymous central defect due to involvement of the posterior angle of the chiasm; *3 C*, homonymous hemianopia with central scotoma due to involvement of the posterior angle; *4 A*, incongruous hemianopic defect; *4 B*, incongruous hemianopic central defects due to involvement of the optic tract; *5*, involvement of the geniculate body; *6 A*, homonymous paracentral scotomas due to rostral involvement of the optic radiation (?); *6 B*, characteristic quadrantic defect due to lesions; *7 A*, bilateral central scotomas due to bilateral posterior occipital lesions; *7 B* and *C*, homonymous congruous defects due to a posterior occipital lesion; *7 D*, defect due to bilateral lesions of the posterior pole and a unilateral lesion of the optic radiation.

9. Rønne, H.: The Different Types of Defects of the Field of Vision, J. A. M. A. **89**:1860-1865 (Nov. 26) 1927.

as "junction scotomata." [10] He remarked that the defect is rarely nasal in the fields. Although such a central field defect may be observed in the field of only one eye, it is probable that in many instances there is a peripheral defect in the field of the other eye as a result of involvement of looped ventral fibers.

In the Optic Chiasm.—1. Lesions at the anterior angle of the chiasm account for field defects which may be similar to those observed as a result of involvement at the termination of the optic nerve.

2. Lesions at the posterior chiasm cause characteristic bitemporal, central and peripheral field defects.

3. Lesions at the posterior angle of the chiasm result in homonymous hemianopia. Involvement of the crossed macular fibers, which enter the root of the tract, may account for loss of visual acuity in the eye on the side of the hemianopia.[11] Again there may be involvement of both the crossed and the uncrossed macular fibers of the eye on the side of the lesion and of the crossed macular fibers of the opposite eye. This gives rise to a pericentral scotoma on the side of the lesion and a temporally placed hemianopic central scotoma in the opposite field. Such field defects indicate involvement of the posterior portion of the chiasm extending into the nerve.

In the Optic Tract.—1. Lesions at the root of the tract are similar to those at the posterior angle of the chiasm.

2. Lesions at the caudal end of the tract are similar to those in the geniculate body.

3. Lesions of the optic tract are characterized by incongruity, probably due to the fact that retinal fibers from homologous points are not here closely associated.

In the Geniculate Body.—The arrangement of the peripheral fibers in the geniculate body accounts for quadrantic hemianopic defects with or without homonymous central defects.

In the Optic Radiation.—Pure quadrantic hemianopia is without doubt the most important single indication of the site of the lesion being in the radiation. Here peripheral or central defects are homonymous and are congruous above the level of the temporal lobe.

In the Occipital Cortex.—Pericentral bilateral scotomas from a lesion of the occipital lobe situated either at the extreme posterior pole or in the posterior parts of the radiation occur but are rarely seen in peace time practice. Holmes' observations made during the World War established that only bilateral lesions accounted for bilateral pericentral

10. Traquair, H. M.: An Introduction to Clinical Perimetry, St. Louis, C. V. Mosby Company, 1927, p. 70.

11. Rønne, H.: The Focal Diagnostic of the Visual Path, Acta ophth. **16**:446-453, 1938.

scotomas. Unilateral lesions caused congruous hemianopic defects which, when quadrantic, extended to the point of fixation.[12] Homonymous hemianopias associated with bilateral central scotomas were observed in several instances.

"Sparing of the Macula."—This feature so frequently considered as indicating a lesion of the optic radiation is probably due in great part to a shift in fixation. In such cases it has been our observation that the blindspot is almost constantly moved inward in the single field in which it can be charted. True "sparing of the macula" undoubtedly occurs in occipital cortical lesions, as is evidenced by the occurrence of bilateral homonymous hemianopia with retention of central visual acuity and small central fields.

CENTRAL SCOTOMAS (CLINICAL CONSIDERATIONS)

It would seem obvious that in considering the diagnostic value of central scotomas, a partial outline, at least of their more common etiologic factors, should be stated. Such an outline is submitted. It is not our purpose to discuss all of the conditions which might account for central scotomas; those due to poisons and toxins are not considered in this paper because they are generally well known.

1. Macular lesions (retinal or choroidal)
   - Inflammatory
   - Degenerative
   - Vascular
2. Inflammations
   - Syphilis
   - Disease of accessory sinuses
   - Arachnoiditis
3. Mechanical
   - Pressure
     - Traction pressure
     - Interference with blood supply
   - Injuries
     - Fractures of base
     - Fractures of optic foramen
   - Tumors
     - Frontal lobe
     - Olfactory groove
     - Suprasellar meningioma
     - Pituitary
     - Hypophysial stalk
   - Aneurysms

12. Holmes, G., and Lister, W. T.: Disturbances of Vision from Cerebral Lesions with Special Reference to the Cortical Representation of the Macula, Brain **39**:34-73, 1916.

4. Demyelinating diseases
   Disseminated sclerosis
   Neuromyelitis optica
   Encephalomyelitis following measles, mumps, chickenpox, vaccinia
   Schilder's disease
5. Poisons and toxins
   Alcohol, tobacco
   Lead
6. Vitamin deficiencies
   Beriberi
   Pellagra
   Tropical vitamin deficiency
   Pernicious anemia (?)
   Diabetes (?)
7. Hereditary degenerations
   Leber's disease
   Hereditary atrophy of the optic nerve

Central Field Defects Due to Macular Lesions.—1. Differentiation Between Interruption of Fibers and Vascular Obstruction: Interruption to nerve fiber bundles usually causes a field defect which is indistinguishable from that of vascular obstruction. Central scotomas arising respectively from a lesion interrupting the nerve fibers and from vascular obstruction can be differentiated, according to Rønne and Traquair. Occasionally in glaucoma only the macular fibers are involved, and in such cases in their early stages the central scotomas "spare the macula," whereas in vascular obstructions there is a "splitting of the macula." We have not observed such cases.

2. Central Scotomas in Maculocerebral Degeneration: Ophthalmologists arrive at this diagnosis by recognizing bilateral macular changes. One patient under our observation exhibited central defects in her visual fields and suffered pronounced reduction in visual acuity for over two years, during which the results of ophthalmoscopic examinations were negative.

CASE 1.—*Macular degeneration in a child 11 years old whose fundi seemed normal during two years of observation, during which time she continued to complain of defective vision; later development of typical changes.*

R. B., aged 11 years, was first seen in October 1935, at which time she gave a history of difficulty in seeing the blackboard for one and one-half years. This difficulty had recently been greatly increased. The family history was noncontributory. She had one brother, aged 16, who was living and well. Both parents had normal vision.

The patient had had measles, chickenpox, scarlet fever and pertussis as a small child. At 15 months of age she had an operation on a gland in the left side of the neck. Tonsillectomy was performed when she was 3½ years of age. At the time of examination she was in the sixth grade, and because of defective vision she had done badly the previous year. Three years before the eyes were

puffed below, and the attending physician found sugar in the urine; the sugar content of the blood was likewise increased. At the time of the examination the blood sugar content was normal. The child had not had any special diet or insulin.

A year and a half before the examination she complained of difficulty in vision, for which glasses did not afford relief. There were no other complaints.

External examination of the eyes showed them to be normal. Vision in the right eye was 20/70-1 and in the left eye 20/100. Retinoscopic examination did not reveal any considerable error in either eye. The results of ophthalmoscopic examination were negative. The visual fields showed a central scotoma in each eye. The peripheral fields were normal.

The possibilities considered at this time were macular degeneration, bilateral retrobulbar neuritis and hysteria.

General physical examination gave entirely negative results. The responses to intelligence tests were constantly above normal level.

During 1936 and 1937 the vision remained at about the same level, and the child was lost from observation from June 1936 until April 1939. At the time of the latter examination the vision was 20/200 in the right eye and 20/70 in the left eye. Bilateral macular changes were now readily visible in each eye. There was no evidence of mental deterioration.

The direct converse of the preceding case has been observed in a Negro child of 5, in whom a bilateral macular change, apparently progressive, had been observed over a period of three months, during which the vision constantly appeared to be normal.

We regard these 2 cases as exceptional.

3. Central Scotoma as a Result of Iridocyclitis: Occasionally it is extremely important to know that a central field defect may arise as a result of iridocyclitis, even though it may be of slight severity. The subject has been reviewed by Østerberg.[13] The central scotoma usually develops insidiously during the long-continued iridocyclitis, sometimes within a month or two and again after a lapse of as long as a year or two. Ophthalmoscopically visible changes may be minimal or absent. Some observers believe that the scotoma is accounted for by macular detachment[14] and others that it is due to retrobulbar neuritis;[15] it has also been suggested that both factors may be present.[16]

4. Central Field Defects Due to "Idiopathic Detachment of the Macula"[17] or "Central Angiospastic Retinopathy":[18] This condition

13. Østerberg, G.: Iridocyclitis with Central Scotoma, Acta ophth. **9**:310-315, 1931.

14. Wolff, E.: Macular Detachment in Iridocyclitis, Proc. Roy. Soc. Med. (Sect. Ophth.) **24**:55-62, 1931.

15. Meller, cited by Østerberg.[13]

16. Clegg, J. G.: Central Scotoma in Anterior Uveitis: A Further Contribution, Tr. Am. Ophth. Soc. **20**:99, 1922.

17. Walsh, F. B., and Sloan, L. L.: Idiopathic Flat Detachment of the Macula, Am. J. Ophth. **19**:195-208, 1936.

18. Gifford, S. R., and Marquardt, G.: Central Angiospastic Retinopathy, Arch. Ophth. **21**:211-228 (Feb.) 1939.

characteristically occurs in high-strung males of 30 to 40 or thereabouts. The central field defect may be either pericentral or paracentral and usually occurs first in the field of one eye and then in that of the other. Central vision is reduced, although not greatly as a rule. Other characteristic symptoms are relative hyperopia, metamorphopsia and micropsia.[19] The diagnosis is made from the symptoms and recognition of a swelling or detachment of the macula with the ophthalmoscope. Unless a binocular instrument is used, the diagnosis is likely to be overlooked. We have not observed syphilitic central retinitis, but apparently it acts in the same manner as this entity.

5. Central Field Defects Due to Strabismus: It is generally accepted that in squinting eyes there is constantly a central defect in the visual field. This may be difficult to demonstrate with ordinary methods of perimetry. Evans,[20] by the technic of angioscotometry, has shown that 100 per cent of these subjects had a demonstrable absolute central scotoma, which, however, was often very small. We have not attempted any careful studies on such scotomas.

Central Scotomas Due to Inflammatory Lesions.—1. Syphilis: Central field changes resulting from syphilis deserve mention. Field defects in cases of parenchymatous and meningovascular syphilis can be considered together, as they are essentially similar.

The presence of a central scotoma is generally considered to indicate that the etiologic factor probably is not syphilis. This is particularly the case if the central scotoma is not associated with a defect of the peripheral field. This point of view has been held since Uhthoff[21] summarized the results of perimetric studies in cases of tabetic atrophy of the optic nerve and stated as his belief that about 2 per cent of central scotomas were caused by syphilis. Fuchs[22] considered this figure too low. Recent studies by Sloan and Woods[23] seem to prove beyond reasonable doubt that central scotomas appear not infrequently in the visual fields of persons with tabes and the tabetic form of dementia paralytica. In a relatively small proportion of their cases the peripheral fields were found to be intact. While it is admitted that exclusion of other factors, such as alcohol and tobacco, which might account for central field defects cannot be made with certainty, in this particular group of patients Sloan and Woods conscientiously endeavored to

19. Walsh and Sloan.[17] Gifford and Marquardt.[18]

20. Evans, J. N.: A Scotoma Associated with Strabismus, Am. J. Ophth. **12**:194, 1929.

21. Uhthoff, W.: Die Augenveränderungen bei Erkrankungen des Nervensystems, in Graefe, A., and Saemisch, E.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1911, vol. 11, pt. 2a, chap. 22, pp. 181-411.

22. Fuchs, E.: The Field of Vision in Tabetic Atrophy of the Optic Disc, Tr. Am. Ophth. Soc. **12**:718-727, 1911.

23. Sloan, L. L., and Woods, A. C.: Perimetric Studies in Syphilitic Optic Neuropathies, Arch. Ophth. **20**:201-253 (Aug.) 1938.

eliminate these factors. They also expressed the belief that homonymous hemianopic field defects in cases of syphilis are usually accounted for by coexistent vascular disease.

Homonymous paracentral scotomas, usually incongruous, are often overlooked because there is no reduction in visual acuity but merely difficulty in reading. Such hemianopic scotomas occur in nonsyphilitic persons as well and have been described by several observers.[24] The exact localization of the lesion accounting for such defects is at present impossible; probably it is in the tract or the rostral portion of the radiation.

2. Diseases of the Accessory Nasal Sinuses: It is our experience that purulent conditions, particularly of the posterior accessory sinuses, sometimes account for meningitis and result in variable field defects, including central scotomas. Nonsuppurative diseases of the accessory sinuses rarely account for retrobulbar neuritis and central scotoma. Gifford's advice to exclude carefully the possibility of tumor of the brain and disseminated sclerosis when sinus disease is suspected of accounting for retrobulbar neuritis continues to be overlooked all too often.

3. Arachnoiditis: We have insufficient experience with this condition to discuss its field defects. In the few cases we have seen in which such a diagnosis was suggested it was not confirmed at operation. Readers are referred to the work of Heuer and Vail,[25] Bollack, David and Puech [26] and Vail.[27]

Central Scotomas from Mechanical Forces.—Pressure, Traction Pressure and Interference with Blood Supply: Under this heading injuries, tumors and aneurysms are included.

Fracture of Optic Foramen: Lillie and Adson [28] reported a valuable localizing sign for fracture of the optic foramen. Their report con-

24. Barkan, O., and Barkan, H.: Central and Paracentral Homonymous Hemianopic Scotomas, Am. J. Ophth. **13**:853-858, 1930. Barkan, O., and Boyle, S. T.: Paracentral Homonymous Hemianopic Scotoma, Arch. Ophth. **14**:957-959 (Dec.) 1935. Allen, T. D., and Carman, H. F., Jr.: Homonymous Hemianopic Paracentral Scotoma, ibid. **20**:846-849 (Nov.) 1938.

25. Heuer, G. J., and Vail, D. T., Jr.: Chronic Cisternal Arachnoiditis Producing Symptoms of Involvement of the Optic Nerves and Chiasm: Pathology and Results of Operative Treatment in Four Cases, Arch. Ophth. **5**:334-349 (March) 1931.

26. Bollack, J.; David, M., and Puech, P.: Les arachnoïdites optochiasmatiques: Etude ophtalmologique et neuro-chirurgicale, Paris, Masson & Cie, 1937.

27. Vail, D. T.: Syphilitic Opticochiasmatic Arachnoiditis, Am. J. Ophth. **22**:505-517, 1939.

28. Lillie, W. I., and Adson, A. W.: Unilateral Central and Annular Scotoma Produced by Callus from Fracture Extending into Optic Canal: Report of Two Cases, Arch. Ophth. **12**:500-508 (Oct.) 1934.

cerned 2 patients who had suffered cranial injury but whose roentgenograms were seemingly negative for fracture. Several weeks after the injury there was pronounced visual loss and the development of a central and a ring scotoma in the visual field. A second roentgenogram of both patients demonstrated the formation of callus, which undoubtedly was responsible for the loss of vision.

Tumors: Tumor of the frontal lobe.[29] Occasionally, although not frequently in our experience, such tumors account for homolateral atrophy of the optic nerve, central scotoma and anosmia. Other tumors in this region, for example meningiomas, also may account for central field defects. In connection with meningiomas, a fact of clinical importance is exemplified by the following case report:

CASE 2.—*Development of a central defect in the left visual field during a late stage of a third pregnancy; sinusitis suspected as the etiologic factor; operative intervention considered contraindicated; recovery of vision within two or three months after delivery; patient remained well for two years; scotoma recurred and persisted for three years, when blurring of vision occurred in the right eye; bitemporal field defects suggested tumor in the region of the chiasm.*

M. K., a white woman aged 38, was seen in August 1936. She complained of blurred vision in the left eye for five years and slight blurring in the right eye for two months. Her family history was unimportant. Her past history was unimportant until five years before examination, when late in her third pregnancy a central scotoma appeared in the field of her left eye. It was promptly atrributed to sinus disease, but operation was thought to be contraindicated. In any event, the field defect disappeared two or three months after the birth of the child. Three years before her examination here it recurred and persisted but had not changed in the six months before I saw her. Two months before examination a blur was noticed in the central visual field of the right eye.

External examination of the eyes gave negative results. Vision in the right eye was 20/30; with a $+0.75$ cyl., axis 90 it was 20/15-2 and the patient could read Jaeger's test type 1 at 9 to 16 inches (23 to 40 cm.). Vision in the left eye was 20/70; with a $+0.50$ sph. $\bigcirc$ $+0.25$ cyl., axis 90 it was 20/40, and the patient could read Jaeger's test type 1 at 9 inches. Ophthalmoscopic examination showed the right nerve and fundus to be apparently normal; in the left nerve head there was barely a suggestion of temporal pallor. The visual fields showed bitemporal defects (fig. 3). Roentgenograms were negative. It was thought that the patient had a tumor in the region of the chiasm.

On October 3 part of the left frontal lobe was resected; a dural endothelioma arising from the olivary eminence was removed. The left optic nerve had to be divided.

After a stormy postoperative course, during which two further resections of the necrotic frontal lobe were performed, the patient recovered. Vision in the right eye was 20/70; with a pinhole it was 20/50. There was no perception of light in the left eye.

29. Voris, H. C.; Adson, A. W., and Moersch, F. P.: Tumors of the Frontal Lobe: Clinical Observations in a Series Verified Microscopically, J. A. M. A. **104**:93-99 (Jan. 12) 1935.

Comment.—Bitemporal field defects probably occurring as a result of physiologic enlargement of the hypophysis during pregnancy have been described frequently. It would seem that often such defects can be classed as functional. That this is not always the case is evidenced by the foregoing case summary.

Suprasellar meningioma. Hagedoorn [30] described a case essentially similar in which pregnancy unquestionably accounted for exacerbation of symptoms produced by a suprasellar meningioma. Fischer [31]

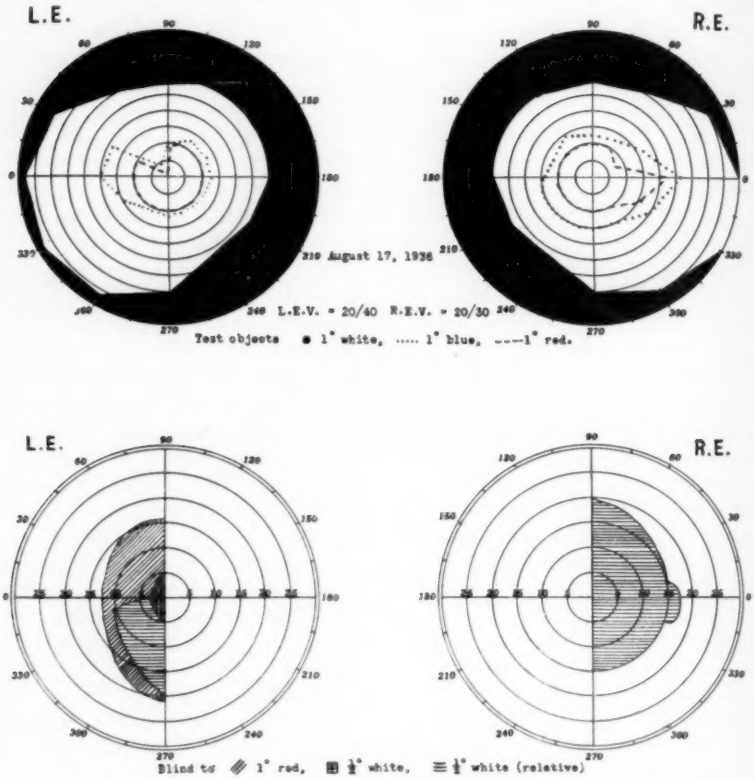

Fig. 3 (case 2).—Peripheral and central visual fields of M. K.

described several such cases in which pregnancy accounted for exacerbation of symptoms in intracranial tumors.

CASE 3.—*Meningioma situated in position of a tumor of the olfactory groove arising from the falx; unilateral visual field defect of the cecocentral type; operation; recovery.*

30. Hagedoorn, A.: The Chiasmal Syndrome and Retrobulbar Neuritis in Pregnancy, Am. J. Ophth. **20**:690-699, 1937.

31. Fischer, cited by Hagedoorn.[30]

R. H., a white man aged 31, was seen in November 1937, complaining of headache and blurring of vision in the left eye. The family and past histories were noncontributory.

For eight months he had severe pains in the vertex of the skull. Pain appeared two or three times a week and lasted from one-half to one hour, occasionally awakening him at night. There were no convulsions, nor was there loss of consciousness or vomiting. A week before examination there was blurring of objects in the field of the left eye. Two days before examination he had had diplopia.

General physical examination gave negative results. Neurologic examination of the cranial nerves was carried out. The patient recognized peppermint, benzine and cloves. He did not show any lesions of the third, fourth and sixth nerves on gross tests. Definite involvement of the fifth nerve was shown by right-sided corneal hypesthesia. There was less marked involvement of facial sensation. There was no motor weakness of the fifth nerve. Auditory acuity was grossly normal. The ninth, tenth, eleventh and twelfth nerves were normal. Motor power was normal. The deep tendon reflexes were sluggish except the right

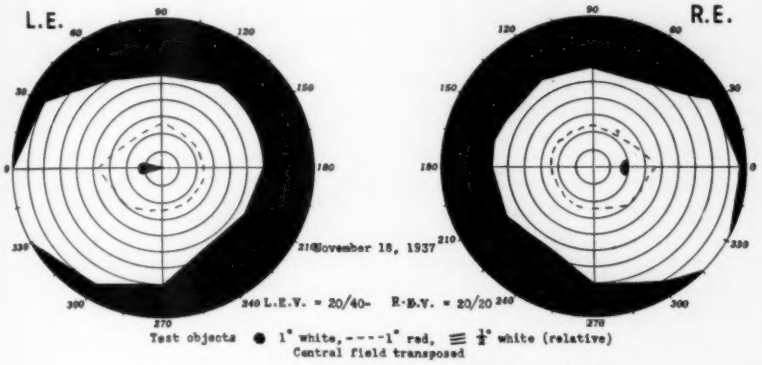

Fig. 4 (case 3).—Peripheral visual fields of R. H.

patellar and the right ankle jerk. Babinski and Oppenheim signs were present on the right. The Gordon sign was negative. Abdominal and cremasteric reflexes were present and equal on the two sides. The sensory responses were normal. There were no signs of cerebellar involvement. Examination for evidence of cerebral involvement showed the patient to be cooperative and cheerful. He was well oriented, and there were no mood disturbances.

Externally the eyes were normal except for a weakness of the right external rectus muscle, demonstrated by diplopia fields. The fields for form and color were normal for both eyes. There was a cecocentral scotoma in the left visual field. Vision in the right eye was 20/20 and in the left eye 20/40. Ophthalmoscopic examination showed pallor of the right disk on the temporal side. The left nerve head was normal. The fundi were otherwise normal.

The usual laboratory tests gave negative results. A roentgenogram of the skull showed destruction of the posterior clinoid processes.

Injection of air was attempted. No air reached the right ventricle. The third ventricle was oblique and dilated, so that the block appeared to be at the aqueduct of Sylvius.

A right craniotomy was performed. The right frontal lobe was resected to expose a tumor in the position of a tumor of the olfactory groove but arising from

the falx. The tumor was in the midline. It weighed 58 Gm. The diagnosis on the basis of the microscopic picture was meningioma.

Comment.—Although the diagnostic signs seemed to point to a left-sided lesion, Dr. Dandy performed a right craniotomy and found the tumor in the midline. The cecocentral scotoma doubtless arose through direct pressure on the left optic nerve. In such cases it would seem, therefore, that this sign should be considered of great value in topical diagnosis.

Pituitary tumor. Before discussing examples of tumors of the intrasellar and hypophysial duct, we should like to call attention to de Schweinitz' classic monograph.[32] In it the ophthalmologic signs of pituitary tumor were so clearly described that little has been added. However, some important questions still remain to be answered. Not the least important gap in the knowledge is the explanation of the field defects in the absence of possible contact between the neoplasm and the optic pathways. Disregard of central field defects and, to a slightly lesser degree, a lack of suspicion of tumor when the nerve heads appear normal account for delay in diagnosis of pituitary tumors. The following 2 cases illustrate the first error.

CASE 4.—*Pituitary tumor; central scotoma and minimal involvement of temporal field of same eye, the only ocular sign; deciding factor in the diagnosis, a roentgenogram of the sella turcica.*

P. G., a white man aged 37, appeared for examination in February 1935. He complained of some dimness of vision of the left eye for two weeks; otherwise there were no complaints of any kind except for occasional headaches, which he had had for many years.

Examination of the eyes gave essentially negative results. Vision in the right eye was 20/15, and the visual field was normal. Vision in the left eye was 20/100. The visual field showed a central scotoma for a ½ degree white test object and colors as well as a peripheral defect for colors. It was suggested that the patient might have an "idiopathic flat detachment of the macula" or a hypertrophic sinusitis. A roentgenogram showed enlargement of the sella turcica downward and forward with thinning suggesting an intrasellar tumor. The sugar tolerance curve was slightly elevated, with a normal sugar level during fasting. The results of all other laboratory tests were negative.

Through a left craniotomy Dr. Dandy removed a chromophobe adenoma which weighed 2.8 Gm. Convalescence was uninteresting. A return visit by the patient in July 1936 showed the vision to be 20/15-1 and 20/15-2, respectively, and the visual fields to be normal.

The following case illustrates the occurrence of tumor in a patient in whom erroneous diagnoses of sinusitis and of arachnoiditis had been made. This case is remarkable particularly from the standpoint of the changing nature of the visual fields.

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32. de Schweinitz, G. E.: Concerning Certain Ocular Aspects of Pituitary Body Disorders, Mainly Exclusive of the Usual Central and Peripheral Hemianopic Field Defects, Tr. Ophth. Soc. U. Kingdom **43**:12-109, 1923.

CASE 5.—*Long-continued persistent headache thought to be due to sinusitis; relief for several months after operation on the sinuses; recurrence of headache; possibility of arachnoiditis suggested by referring physician; final diagnosis, pituitary tumor; operation; recovery; changeable nature of visual fields the interesting feature; bitemporal hemianopic central scotoma observed early in the course of the disease, then an apparent homonymous hemianopia and finally a bitemporal hemianopic field defect.*

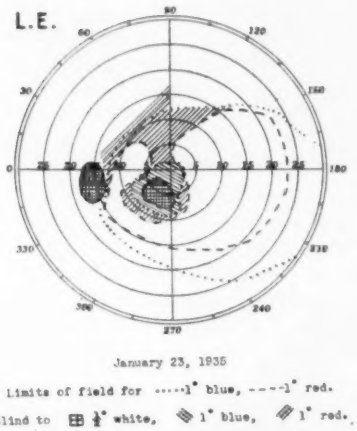

Fig. 5 (case 4).—Peripheral and central visual fields of P. G. The test was incomplete. The results with the 1 degree blue and red test objects indicated that there was a peripheral field defect also in the temporal field.

B. H., a white man aged 45, was first seen on March 22, 1939. He complained of blurring of vision and inability to see to the right. The family history was noncontributory. According to the past history, the patient had good health until the onset of the present illness.

The present illness began four years before examination. The patient began to have intermittent, sharp shooting pains in the right side of the forehead which seemed to begin in the region of the right frontal sinus and radiate to the right

vertex. At first the pains were infrequent, but they became more severe during March 1938. At this time exploration of the right frontal sinus was done, and it was reported that polypi and pus were removed therefrom. Pigmented moles were said to have been removed from the back and from the right upper eyelid. After exploration of the sinuses, the patient was apparently relieved for eight months. Four months before examination (November 1938) he began to notice bilateral diminution of vision. In January 1939 he was unable to see to the right, and at the same time he was conscious of a blindspot in the left eye. He started to have a generalized headache, which, however, was not associated with nausea or vomiting. An encephalogram had been made, but it was reported as negative. The possibility of arachnoiditis was suggested by the referring physician.

General physical examination gave negative results. A search was made for pigmented moles, but none were found. Neurologic examination gave negative results.

Examination of the eyes showed them to be normal externally and on ophthalmoscopic examination. Vision in the right eye was 20/30; with a + 0.50 cyl., axis 90 it was 20/20. Vision in the left eye was 20/20. The visual fields showed right homonymous hemianopia. Previous and subsequent fields are shown in figures 6 and 7.

Injection of air was performed. The ventricles were symmetric and appeared normal, except that the temporal horn of the left lateral ventricle was pushed backward just at its tip. From the history of moles it was suspected that the patient might have a metastatic melanotic tumor.

On March 25 a hypophysial approach was made on the left side. The carotid artery was found to be pushed aside by a mass situated between the posterior border of the nerve and the artery. This mass was a darkish red. It was debated whether it was a melanotic tumor or an aneurysm. The fact that blood could be drawn from it with a syringe pointed strongly to the latter diagnosis. A clip was applied to the carotid artery just before its bifurcation. On April 10 the patient was discharged from the hospital. He continued to complain of the visual defect.

In October he was readmitted to the hospital. He continued to complain of pain in the right side of the head and felt that there had been some reduction of vision. Vision in the right eye was 20/40; with a + 0.50 cyl., axis 90 it was 20/30-2. Vision in the left eye was 20/100, eccentric. Ophthalmoscopic examination gave negative results. The fields showed bitemporal hemianopia.

On November 1 a left craniotomy was done. The left optic nerve was divided, and a bulging mass was found in the hypophysial region. The tumor, an adenoma, was removed except for a small portion of its capsule. Vision in the right eye on discharge was 20/15-4.

Tumor of the hypophysial stalk. The following case history is of interest from the standpoint of its demonstrating a homonymous hemianopic defect in the visual fields of a patient suffering from a tumor of the hypophysial stalk. In addition, interpretation of the fields was difficult because of the presence of a central scotoma in the right eye due to an old injury.

CASE 6.—*Diabetes insipidus associated with recent visual failure; pronounced reduction of vision and a central scotoma accounted for by an old central lesion in the right eye; vision in the left eye reduced to 20/100; a cecocentral scotoma in the visual field; vision improved in the left eye to 20/15 after operation; recurrence*

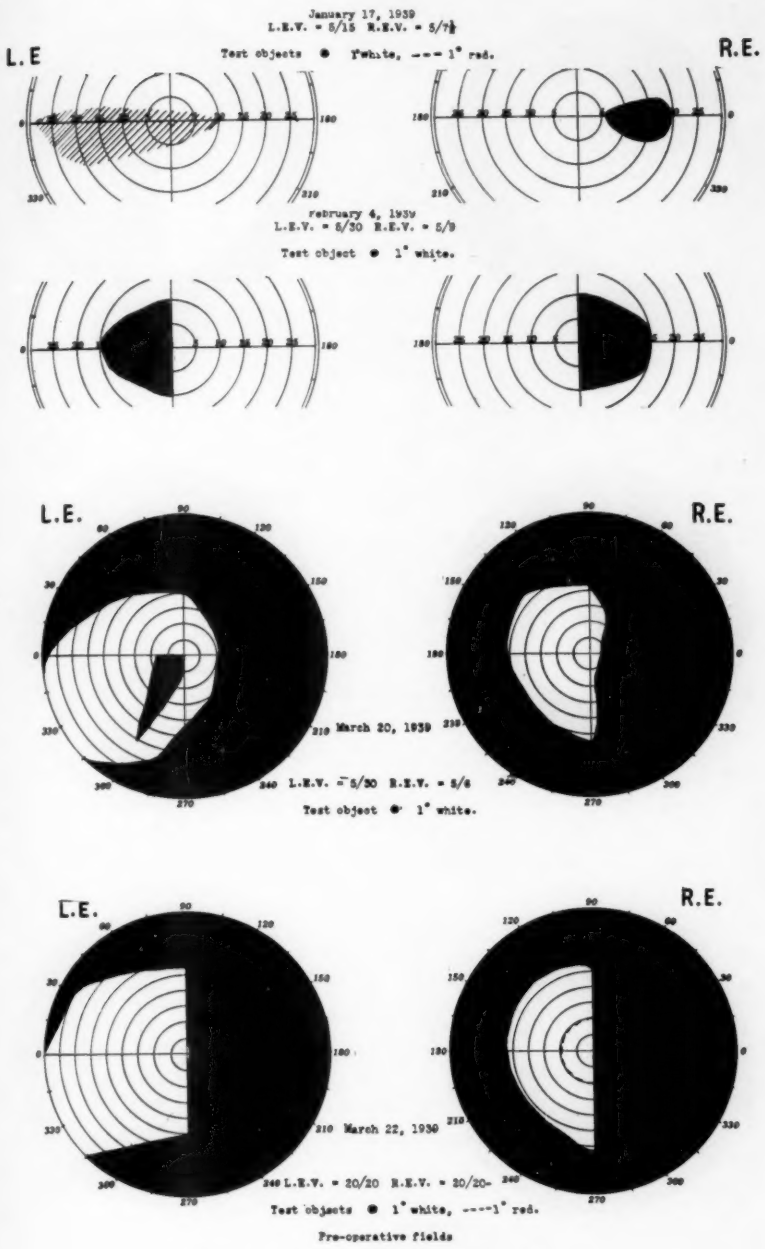

Fig. 6 (case 5).—Visual fields of B. H.

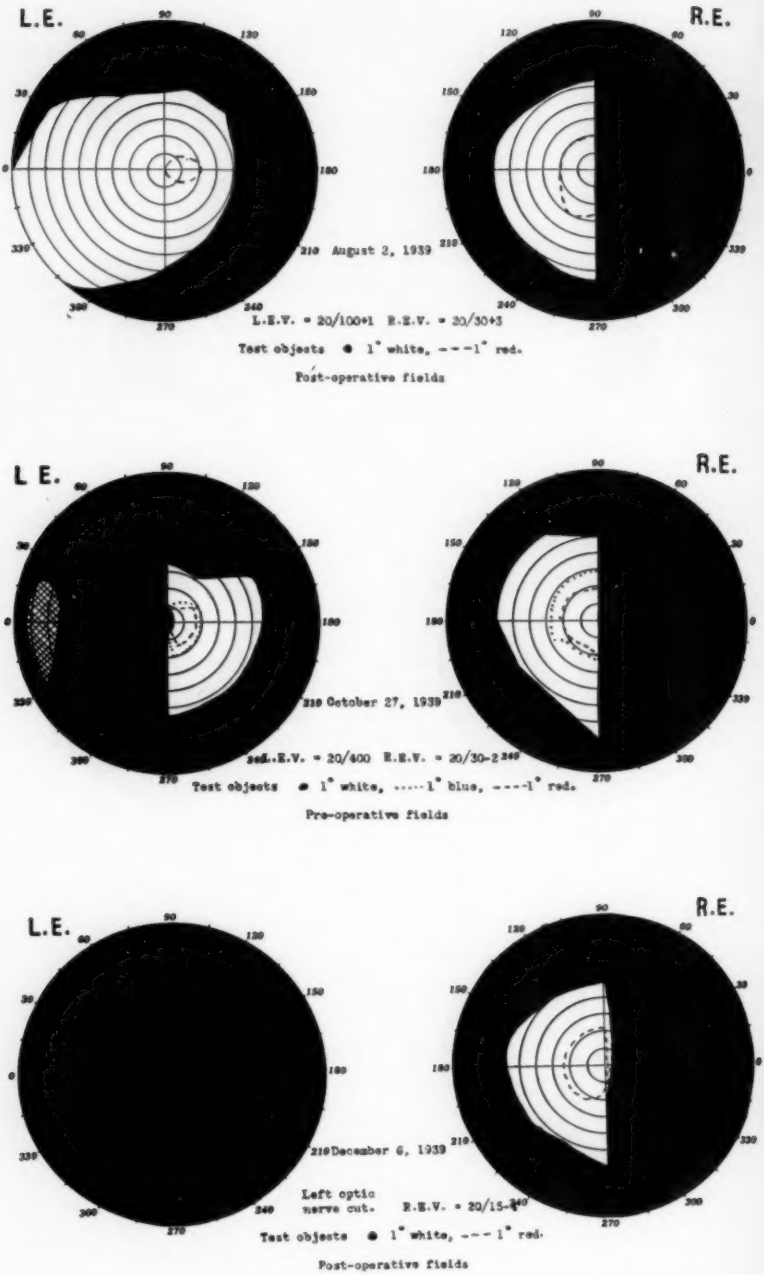

Fig. 7 (case 5).—Visual fields of B. H.

of the tumor, an epidermal carcinoma; right nerve cut, even though vision in the left eye was 1/200 and in the right eye 20/100; recovery of vision to 20/30 in the left eye; recurrence (?) and death.

F. B., a white woman aged 39, was first seen in the Johns Hopkins Hospital in October 1936. About June 1936, she complained of excessive consumption of water and polyuria; up to 13 quarts (12 liters) of water were taken in twenty-four hours. Along with these symptoms, pronounced psychologic instability, crying, depression, generalized nervousness and irritability were described. One cubic centimeter of posterior pituitary extract twice daily cut down her fluid intake

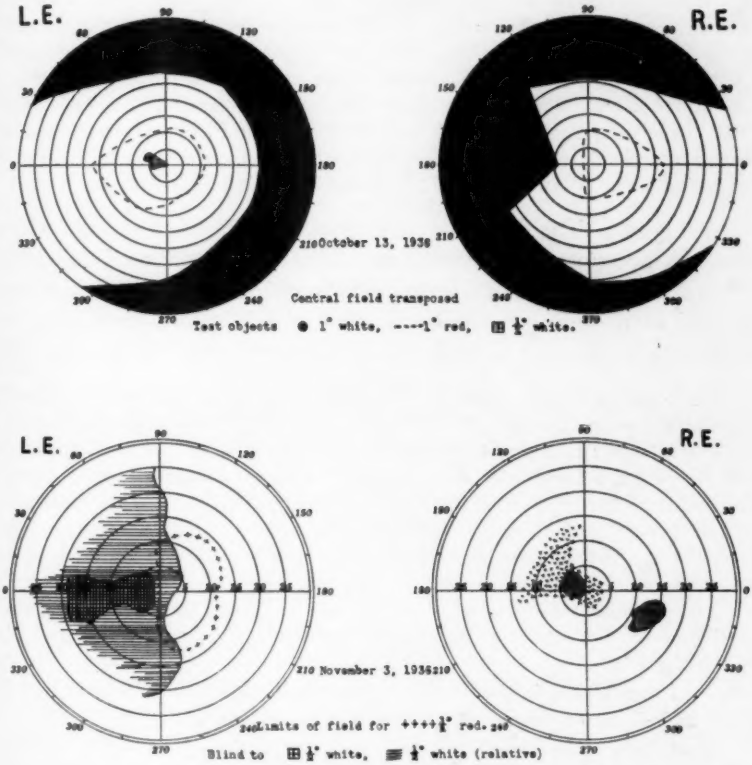

Fig. 8 (case 6).—Peripheral and central visual fields of F. B.

but was discontinued because boils developed. Her previous history was interesting in one detail; nine years before examination the right eye had been injured, and there had been reduced vision with central scotoma since the accident to the eye. For two weeks before entrance to the hospital she had complained of blurring in the center of the left visual field.

Examination of the eyes showed them to be normal externally. Vision in the right eye was 20/200 (eccentric due to old chorioretinal lesion). Vision in the left eye was 20/100. There was some pallor of the temporal side of the right nerve head, but the left ocular fundus was normal. The fields showed essentially homonymous defects.

Roentgenograms of the skull were negative. Injection of air spinally did not aid in the diagnosis.

On November 18 operation was performed by Dr. Dandy with a right hypophysial approach. An ependymal cyst of the hypophysial stalk was discovered below the chiasm. It was punctured, and tenacious fluid was evacuated. The patient recovered.

In December 1936 vision in the right eye was 20/100 and in the left eye 20/15. The field of the left eye was normal. During January the vision rapidly decreased, and the patient was readmitted to the hospital on Feb. 8, 1937. Vision in the right eye was 20/100-1 and in the left eye 1/200.

A second operation was performed on February 12. At this operation it was found necessary to sever one optic nerve, because there was a central lesion in the right eye, although at the time the vision was as stated. The tumor was found to have recurred. After the operation, there was partial paralysis of the right third nerve and paralysis of the right sixth nerve; there was also a hemianopic defect in the field of the left eye. On March 3 vision in the left eye was 20/30—surely a remarkable recovery. Unfortunately there was rapid return of the growth. The patient died on April 28, 1937. Autopsy was not performed.

Aneurysm: The following case demonstrates misdiagnosis when an aneurysm accounted for symptoms which were attributed to sinusitis. Unfortunately the patient's severe illness and lack of cooperation caused difficulty in obtaining a reliable history and in taking the fields, when the diagnosis depended in great part on interpretation of the visual fields.

CASE 7.—*Aneurysm of anterior communicating artery; long-continued persistent headache, defective vision, photophobia and diplopia; operation on sinuses failed to relieve symptoms; vascular hypertension, mental confusion continuous low grade fever and probable coarctation of aorta found on examination; roentgen evidence of involvement of posterior sinuses; ill defined temporal field defects; reoperation on posterior sinuses and evacuation of sterile pus, without relief; aneurysm revealed by craniotomy but ligature of carotid artery contraindicated; death from spontaneous rupture three months later.*

W. G., a white man aged 41, was first seen on Jan. 11, 1939, complaining of defective vision in both eyes, pain in the eyes on exposure to light and pain in the frontal regions.

He had been kicked in the head by a mule at the age of 10. In 1920 and again in 1937 he had been hospitalized for vascular hypertension.

In April 1938 he had been having headaches several weeks, when he had a convulsion which lasted for twenty minutes. He was admitted to a hospital for study. Examinations included two lumbar punctures. No diagnosis was made other than vascular hypertension. In October 1938 an operation was performed on the sinuses. The headaches continued.

At the time of his admission to the Johns Hopkins Hospital (Jan. 11, 1939) he complained of defective vision in both eyes which had been present for eight months, double vision for two months and photophobia for three months. There was also complaint of dyspnea on exertion and an indefinite history of pains in the precordium extending down the left arm. There were irregular periods of "roaring" in the right ear.

The temperature was moderately elevated. The patient was a well developed man of 40. He was uncooperative and complained that light hurt his eyes. The head was normal. There was no audible bruit over the skull or eyes. No tender-

ness was elicited over the bones, sinuses or mastoid. McEwen's sign was negative. Examination of the nose, throat and teeth did not reveal anything remarkable. The thorax expanded normally. The lungs seemed normal. The heart was not enlarged to percussion. Sounds were normal except for well localized harsh short systolic murmur over the pulmonic area. The abdomen was normal. Inability to obtain blood pressure readings in the legs suggested possibility of coarctation of the aorta.

Neurologic examination gave negative results except for the ophthalmologic findings.

Externally the eyes appeared normal except for redness of the conjunctiva and a slight irregular dilatation of the right pupil. Vision in the right eye was 20/50-1; with correction it was 20/40. Vision in the left eye was 10/200 and could not be improved. The nerve heads were slightly pale; the vessels were normal. A series of fields were charted. In the first field there was a paracentral temporal scotoma in each eye. In subsequent fields there were irregular temporal cuts.

Roentgenograms of the skull showed dense clouding of the sphenoid sinuses with suggestion of destruction of the left clinoid process. A roentgenogram of the chest showed notching of the posterior ribs characteristic of coarctation of the aorta. Serologic tests of the blood and spinal fluid were negative for syphilis. The spinal fluid was clear.

The patient had fever, the temperature fluctuating up to 102 F. The leukocyte count varied between 7 and 10,000. The patient was constantly drowsy. Possibilities considered were recurrence of sinusitis and some lesion about the chiasm. On January 21 the sphenoid sinuses were opened. The right sphenoid sinus was found filled with creamy pus; the left contained serosanguineous material. The pus was sterile on culture. Symptoms persisted and became more pronounced. On Febuary 8 a left craniotomy was performed by Dr. Dandy. A pink encapsulated mass thought to be an aneurysm projected between the chiasm and the optic nerves, pushing the whole chiasm upward. An attempt was made to produce thrombosis by passing a needle through the mass, applying a cautery to the needle. The carotid artery was not ligated because a previous test of compression of the artery in the neck had produced dizziness in less than thirty seconds. At operation the left optic nerve was cut in order to obtain a better view of the aneurysm.

After the operation, the patient remained stuporous for a time but steadily improved, although not recovering completely. On April 8 he suddenly had a generalized convulsion. Spinal puncture revealed grossly bloody fluid, and the patient died a few hours later.

At autopsy it was shown that the aneurysm had ruptured. It was about 2 cm. in diameter and was attached to the anterior communicating artery almost exactly in the midline. It compressed the chiasm and the hypothalamus. It was thought that pressure on the hypothalamus probably accounted for the mental state and increased temperature.

CASE 7a.—*Sudden unilateral loss of vision the only sign of aneurysm of the posterior communicating artery; after several weeks, onset of severe pains in the side of the head; occurrence of subarachnoid hemorrhage; ligation of internal carotid artery followed by contralateral flaccid hemiplegia; hyperpyrexia and death; autopsy evidence of ruptured intracranial aneurysm.*

J. J., a Negro aged 39, was first seen on Jan. 4, 1940, complaining of blindness of the right eye. On Dec. 31, 1939 he first noticed defective vision in the right eye. There were no other complaints. The defect grew rapidly more pronounced. The patient became completely blind in that eye within four days. He had con-

sumed a quart of cheap whisky on the night preceding the onset of blindness, but he had had an average consumption of approximately this amount daily for several months.

Examination of the right eye showed it to be blind. The pupil was dilated. It did not react to direct light stimulation but reacted to consensual stimulation. The nerve head seemed normal. The left eye was normal, with vision of 20/15 and normal fields. A diagnosis of retrobulbar neuritis was suggested, but the cause was not clear. General physical examination gave essentially negative results save for slight vascular hypertension, 150 systolic and 100 diastolic, and a blowing systolic murmur at the apex. A Wassermann test of the blood was positive. Roentgenograms of the skull were normal.

The patient disappeared from observation for several weeks until March 27, when he was brought to the hospital in a semicomatose state. For more than a month there had been a complaint of severe pains in the right side of the head. On the morning of admission he had suffered a sudden excruciating pain in the occipital region and rapidly became semiconscious. Examination showed temperature 99 F., pulse rate 98 and respiratory rate 24; the neck was rigid. The patient was nonresponsive save to painful stimuli, and his combativeness made examination difficult. Both pupils were fixed to light and were dilated. The right optic nerve head was chalky white; the left was normal. There were slight ptosis of the right upper lid and external deviation of that eye. A spinal tap revealed a grossly bloody fluid under increased pressure. The Wassermann test was strongly positive. The condition rapidly became worse. The history seemed to indicate clearly that the subarachnoid hemorhage originated in a ruptured aneurysm on the right. It was decided to operate.

Pressure was applied to the right internal carotid artery for ten minutes, and there was no evidence of subjective change. The operator (Dr. Pickrell) then ligated the artery. Eighteen hours later a flaccid hemorrhage on the left side was found to have developed. Hyperpyrexia manifested itself on the following day. Autopsy revealed a ruptured aneurysm on the right posterior communicating artery and extensive subarachnoid hemorrhage.

Lesions Posterior to the Geniculate Body: Homonymous central field defects are encountered infrequently when such lesions are present.

CASE 8.—*Malignant tumor of the skull extending internally, involving the right occipital lobe; left homonymous hemianopia and bilateral papilledema the only definite signs; symptoms relieved temporarily on removal of tumor compressing the posterior portion of the right occipital lobe; improvement in fields which showed incongruous homonymous hemianopic central scotomas; complete hemianopia with sparing of the macula produced by later recurrence; further operation (1933), consisting of removal of recurrent tumor (66.5 Gm.), a hemangiosarcoma; later (1934) evacuation of a cyst; apparent recovery.*

A. McD., a white woman aged 55, first seen on Feb. 2, 1931, complained of pain in the back of her head for a year. Her family history was noncontributory.

In October 1930 she had had a cyst removed from the right occipitoparietal region. The operative wound healed, but the scar became swollen and painful. Her gait became slightly unsteady.

A year prior to examination she commenced having severe nocturnal headaches, which were situated principally in the occipital region. These would awaken her at night. They occurred several times a week and gradually increased in severity and frequency. There was no nausea or vomiting. From September

1930 to the date of examination the headaches were constant. Pain in the occipital region radiated to the right side of the head. For three months she had complained of loss of memory and had noticed that she occasionally used the wrong words. She had observed defective vision to the left but had not complained of diplopia.

Examination showed an overweight woman of 55, intelligent and cooperative. There was a swelling of the skull about 6 cm. in diameter in the right occipitoparietal area posteriorly, with an old operative scar over the swelling. General physical examination gave negative results. The cranial nerves, except for the second, seemed normal. The motor power was normal. There were no abnormal sensory reactions. The deep reflexes were equal on the two sides and hyperactive. The abdominal reflexes were sluggish. There was no apparent aphasia, apraxia or astereognosis. There were no definite cerebellar signs.

Examination of the eyes showed them to be normal externally. There was bilateral papilledema of 1.5 to 2 diopters in each eye. The peripheral fundi seemed normal. Vision was 20/20 in each eye. The visual fields showed a left homonymous hemianopia.

On February 4 Dr. Dandy operated. An opening in the bone was discovered in the posterior part of the incision in the occipital region. Through this opening the tumor protruded into the brain, pushing the occipital lobe anteriorly and outward. It was a well encapsulated, nodular reddish brown tumor. It extended forward a considerable distance into the occipital lobe and shelled out perfectly. On section it was reported to be a dural endothelioma.

On Jan. 17, 1932 the patient again complained of severe constant pain in the right side of the head. A fluctuant swelling had appeared under the operative wound. The papilledema had disappeared. Vision remained normal. The visual fields now showed paracentral homonymous central defects, absolute for a 1 degree white test object. A repair operation was performed.

The patient was again seen in November 1933, at which time she complained of severe pain and was obviously in acute distress. There was intense bulging in the area of the previous operative wound. There was a questionable Romberg sign. The fields showed a left homonymous hemianopia with sparing of the macula for form and for colors. There was slight papilledema, particularly in the right eye, with a suggestion of a low grade atrophy of the optic nerve.

A further operation was performed (Dr. Dandy) on November 22, when a tumor weighing 66.5 Gm. was removed from the right occipital lobe. Section showed this to be hemangiosarcoma of the brain; the dura showed bone formation.

The patient returned in October 1934, complaining of abdominal pains. She had also observed enlargement of cranial defect but had had only an occasional headache. The eyes were unchanged. A large extraventricular cyst was evacuated. There was communication between the cyst and the lateral ventricle, which was small. Some of the choroid plexus was removed. At this writing there has been no recurrence of the tumor.

Central Scotomas from Demyelinating Diseases.—Central field defects are frequently found as the result of demyelination. Abrupt onset of visual defect is the outstanding and characteristic ocular sign in the demyelinating diseases. This process, however, may or may not spare the optic pathways.

Disseminated Sclerosis: Acute unilateral retrobulbar neuritis is generally considered one of the earliest signs of disseminated sclerosis.

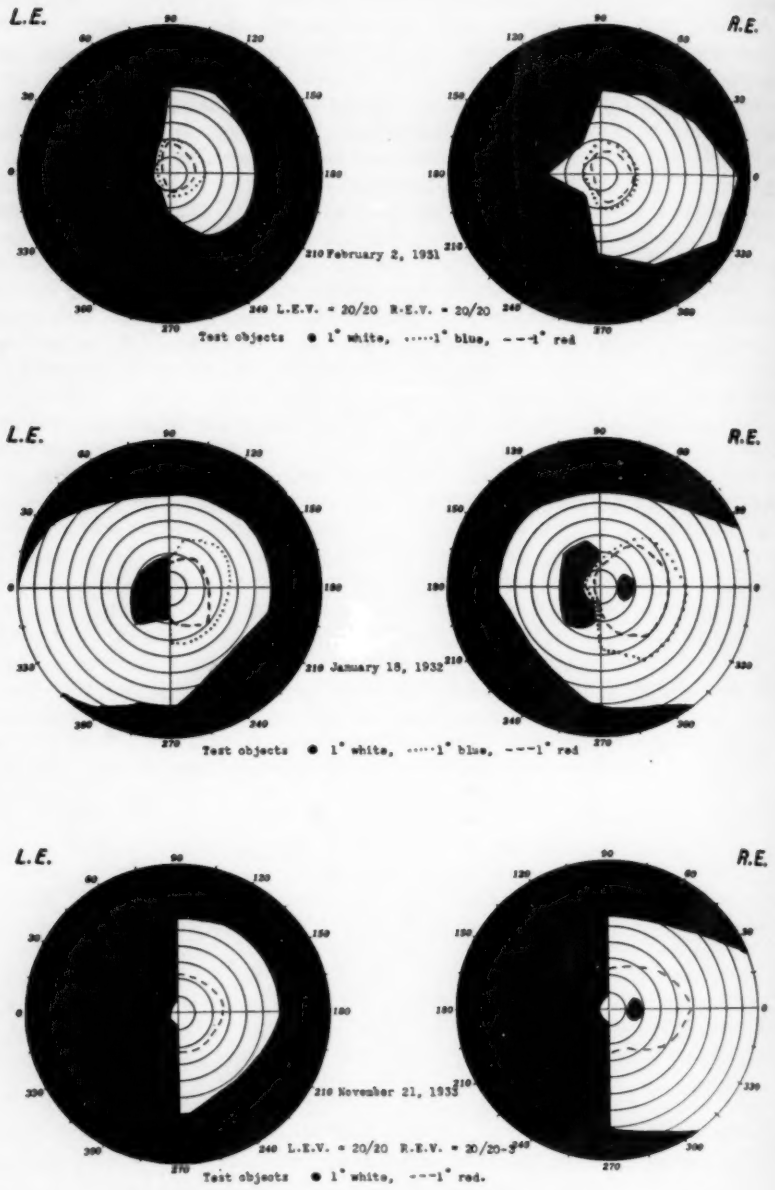
L.E.
R.E.
February 2, 1931
L.E.V. = 20/20 R.E.V. = 20/20
Test objects ● 1° white, ·····1° blue, – – –1° red
L.E.
R.E.
January 18, 1932
Test objects ● 1° white, ·····1° blue, – – –1° red
L.E.
R.E.
November 21, 1933
L.E.V. = 20/20 R.E.V. = 20/20-3
Test objects ● 1° white, – – –1° red.

Fig. 9 (case 8).—Visual fields of A. McD.

This type of retrobulbar neuritis is characterized by rapid onset of visual loss and by a central field defect, usually in the form of a pericentral scotoma. The process is usually transient and frequently recurrent.

Chiasmal involvement in disseminated sclerosis, although infrequent, accounts for field defects which are indistinguishable from those caused by other diseases of the chiasm; consequently the diagnosis of disseminated sclerosis in such cases depends on correct interpretation of available information. Rapid onset of visual loss is always suggestive. Sudden changes in the field defects are commonly seen.

Retrochiasmal involvement is even rarer than chiasmal involvement. Homonymous defects characterize such cases. The involvement appears to be in the tract.

Neuromyelitis Optica: The ocular signs of this disease are unique. There is rapid and bilateral loss of vision which, in some instances, is followed by paraplegia; in other cases paraplegia may precede the ocular symptoms. Whether or not ocular signs alone should lead to a diagnosis of neuromyelitis optica is an open question. Certainly it is not uncommon to observe patients with typical ocular signs in whom myelitis fails to develop within periods of observation of several years. The significance of bilateral rapid loss of vision as indicating neuromyelitis optica does not seem to be generally appreciated. Confusion between this disease and tumor of the brain would be avoided if it were recognized that bilateral loss of vision progressing rapidly to blindness or near blindness is typical of neuromyelitis optica and is not seen in tumor of the brain.

Whether or not neuromyelitis optica is an acute form of disseminated sclerosis raises differences of opinion. In our opinion it can be differentiated clinically from disseminated sclerosis by (1) the extreme youth of some of the patients, (2) the occurrence of bilateral optic neuritis with myelitis and (3) the occurrence of bilateral blindness of far greater severity than that in disseminated sclerosis, in which it is unusual for an eye to become totally blind suddenly and bilateral blindness is even rarer. The chief pathologic differences are the more destructive character of the process resulting in cavity formation and the predilection for involvement of optic nerves and cord in neuromyelitis optica.

The field defects in neuromyelitis optica are variable because the areas of demyelination are irregularly arranged. Central scotomas are frequently present. Often it is impossible to chart the fields because of the patient's critical condition.

Encephalomyelitis: Central field defects may arise as a result of the demyelinating lesions of encephalomyelitis following measles, German measles, vaccination against smallpox or rabies, smallpox, chickenpox, whooping cough and obscure febrile illnesses. When loss of vision is

observed in these cases it is usually bilateral and often extreme; but there is almost always remarkable recovery, with, however, varying grades of atrophy of the optic nerve. It has been established that the severity of the exanthem is not a factor determining the occurrence of encephalomyelitis.

CASE 9.—*Optic neuritis as a single manifestation of encephalomyelitis following chickenpox; vision reduced to perception of light within twenty-four hours; complete recovery.*

F. S., a white boy 7 years of age, was admitted to the hospital on Feb. 17, 1932, complaining of loss of vision which had come on the preceding day. He had been in bed for several days suffering from chickenpox, which had erupted two weeks previously. On the day that he got up he complained of sudden loss of vision.

General physical examination revealed several lesions on the skin compatible with a subsiding attack of chickenpox. Otherwise the physical examination gave negative results. Neurologic examination, except for the ocular examination, gave negative results. Examination of the eyes revealed widely dilated pupils which reacted slightly, if at all, to light. It was doubtful whether or not there was even perception of light. There was bilateral papilledema of 3 to 4 diopters. A diagnosis of retrobulbar neuritis was made. Spinal fluid was normal and contained four cells. Injection of air was performed with negative results. On March 2 vision in the right eye was 20/50 and in the left eye 20/70. The papilledema was receding. On April 4 vision in the right eye was 20/30 + 2 and in the left eye 20/50. On March 21, 1936, vision in each eye was 20/20.

CASE 10.—*Encephalomyelitis following measles.*

J. H., a white boy of 8 years, had measles on June 2, 1935. About June 9 he complained of headaches, difficulty in vision and the hearing of voices, as if he were delirious. The vision failed rapidly, until on July 6 vision in the right eye was 1/500 and in the left eye 5/500. Ophthalmoscopic examination showed some blurring of the margins of the disks. It was impossible to chart the visual field of the right eye. There was a large central scotoma in the left field. The peripheral limits were normal. On July 9 vision in the right eye was 20/40 and in the left eye 20/20. A small central scotoma was charted in the visual field of the right eye. Neurologic examination gave negative results.

Schilder's Disease: Cortical blindness has been described as an outstanding sign in Schilder's disease, but we did not observe it in several patients in whom the presence of the disease was later established at autopsy. Indeed, it is only at autopsy that Schilder's disease is usually recognized as such. Certainly there is nothing characteristic about the ocular signs, including the perimetric findings, unless it is cortical blindness. The fact that the demyelinating lesions may appear in any part of the white matter [33] accounts for the wide variation in symptoms. Loss of vision is a constant finding in the last stages of the disease, but charting of the fields is impossible at this stage. The patient in the following case presented the outstanding histologic char-

33. Ford, F. R., and Bumstead, J. H.: Encephalitis Periaxialis Diffusa of Schilder: Report of Two Cases with Anatomical Findings, Bull. Johns Hopkins Hosp. **44**:443-485, 1929.

acteristics of Schilder's disease, namely, involvement of the white matter and sparing of the gray matter with intact arcuate fibers separating the involved from the uninvolved areas. Involvement of the spinal cord was present, an unusual finding in Schilder's disease.

CASE 11.—*Sudden loss of vision and a large central scotoma in the visual fields; death within a month after first examination; diagnosis of Schilder's disease apparently substantiated by autopsy.*

M. L., a Negress aged 24, was first seen on April 24, 1935. She complained of headache and blindness. The family history was noncontributory. The past history revealed that the patient had always been healthy.

A month before examination the patient began to complain of bifrontal headaches. These continued to become more severe. About two weeks before admission she experienced sudden blurring of vision in the left eye, and two days later it was totally blind. On the following day the left eye became affected, and within two days it became totally blind. For several days before examination she had had complete loss of vision, even down to loss of perception of light. There was then slight improvement.

Temperature, pulse and respirations were normal. Physical examination showed a woman who was definitely euphoric but was well oriented as to time and place and appeared to have a normal level of intelligence. There were generally hyperactive reflexes except for the abdominal reflexes, which were missing on each side. Examination of the eyes showed the pupils to be widely dilated and with only a slight reaction to light. Ophthalmoscopic examination showed blurring of the margins of the disks with a slight amount of edema about them. A small retinal hemorrhage was seen in the inferior quadrant of the left eye. Serologic tests of the blood and spinal fluid were negative for syphilis. The spinal fluid was under normal pressure; there were 10 cells, the Pandy reaction was 2 +, there was 50 mg. of protein per hundred cubic centimeters, and the colloidal mastic curve was 4432110000. Roentgenograms of the skull and sella were negative.

The patient was admitted to the hospital on May 21, at which time she was dying. The respirations were strikingly irregular and at times noisy. She was having tonic spasms of the arms. The right pupil was widely dilated and the right upper lid drooped. The left pupil was very small, not more than 1 mm. in diameter. Neither pupil reacted to light. The right optic nerve showed definite temporal pallor. The left disk was not seen. The patient could not swallow, and foamy saliva flowed from her mouth. The tendon reflexes were everywhere grossly exaggerated. Babinski and Hoffmann signs were bilaterally present. Clonus was present, but the abdominal reflexes were absent. It was thought that the patient had extensive destructive lesions, some of which were located in the optic nerves. Schilder's disease was suspected. She died on the day after admission.

Autopsy showed large areas of demyelination widely scattered throughout the white matter of the brain and a number of similar minute lesions in the spinal cord. The lesions had a perivascular distribution. There was widespread demyelination in the optic nerves. Complete lack of glial proliferation indicated the process was an early and an acute one. Sparing of the arcuate fibers seemed to warrant the diagnosis of Schilder's disease.

Central Scotomas from Vitamin Deficiencies.—Vitamin deficiency plays an important role in the production of retrobulbar neuritis. In this country outspoken vitamin deficiency is observed in cases of the

alcoholic type of pellagra and possibly in cases of chronic alcoholism. It seems probable that relative deficiencies may account for retrobulbar neuritis occasionally observed in cases of pernicious anemia and of diabetes.

Beriberi: The part that vitamin deficiency plays in the production of this disease has been amply proved. Mention of it is made here only because of observations made in cases of beriberi which are of interest in their relation to two conditions already discussed in this paper, namely, central field defects associated with pregnancy and with "idiopathic flat detachment of the macula." Schimazono [34] stated that whereas retrobulbar neuritis was accounted for in Europe by disseminated sclerosis and excessive use of alcohol and tobacco, in Japan it was due to beriberi or to lactation. Ordinarily beriberi is seen most frequently in men; but of 90 patients with retrobulbar neuritis, 30 were men and 60 women, of whom the majority were lactating. Oguchi [35] raised a question as to whether in beriberi there was loss of vision from retrobulbar neuritis or from central retinitis.

Pellagra: Amblyopia in cases of pellagra is properly attributed to deficiency of vitamin B and is usually seen associated with alcoholism. This type of amblyopia has been adequately described by Carroll.[36] We have observed a central scotoma in the fields of a pellagrin of 18 years in whom it seemed unlikely that alcohol played any part. Fine and Lachman [37] stated that ocular symptoms are much less frequent in pellagrins today than they were years ago; they cited in support of this statement Whaley's report [38] in which it was stated that of 35 pellagrins, 3 had optic neuritis, 3 had optic atrophy and 2 had retinitis.

A further point regarding alcoholic amblyopia is that we have observed patients who presented typical symptoms of amblyopia from a single overindulgence, and consequently vitamin deficiency is obviously not the only factor. Such cases are described by Carroll and Goodhart.[39]

Tropical Vitamin Deficiency: Many reports are available in journals of tropical medicine in which epidemics are described as due to vitamin

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34. Shimazono, J.: B-Avitaminosis und Beriberi. Mit Bemerkungen zur alimentären Anämie, Veränderung der Nervensubstanz durch Aufbrauch und zur zentralen Wirkung der Schilddrüsen-substanz, Ergebn. d. inn. Med. u. Kinderh. **39**:1-68, 1931.

35. Oguchi, C.: Ueber das Wesen der Neuritis optica axialis, Acta Soc. ophth. japon. **34**:125-126, 1930.

36. Carroll, F. D.: "Alcohol" Amblyopia, Pellagra, Polyneuritis: Report of Ten Cases, Arch. Ophth. **16**:919-926 (Dec.) 1936.

37. Fine, M., and Lachman, G. S.: Retrobulbar Neuritis in Pellagra, Am. J. Ophth. **20**:708-714, 1937.

38. Whaley, E. M.: Eye Symptoms of Pellagra, Tr. Nat. Conf. on Pellagra, 1909, p. 279; cited by Fine and Lachman.[37]

39. Carroll, F. D., and Goodhart, R.: Acute Alcoholic Amaurosis, Arch. Ophth. **20**:797-803 (Nov.) 1938.

deficiency. The descriptions of the symptoms are essentially similar in many reports from widely separated countries. The patients suffer progressively from sore tongue, sore mouth and white patches on the lips, lesions on the genitals, numbness of the legs and feet and finally reduction of vision. The loss of vision is pronounced but is not complete; there usually is a central field defect rather than peripheral constriction. Atrophy of the optic nerve is seen in cases of advanced and long-standing involvement. Almost all patients with this form of avitaminosis are, if seen reasonably early, rapidly improved by the use of various vitamin B products.[40]

Pernicious Anemia: The frequency of sore tongue as a symptom in pernicious anemia is of interest when it is recalled that it is the outstanding early symptom of "tropical" avitaminosis. It is not generally known that retrobulbar neuritis may account for central field defects as an early symptom of pernicious anemia. Such cases have been recorded by Box [41] and by Kampmeir and Jones.[41a] The infrequency of ocular signs in cases of pernicious anemia is striking, particularly when it is remembered that the involvement of the nervous system often causes confusion between pernicious anemia and tabes dorsalis. Although it has been shown that ordinarily the use of additional vitamin therapy does not enhance liver therapy in pernicous anemia, it might well be given in the rare cases in which retrobulbar neuritis is present.

Diabetes: Occasionally retrobulbar neuritis is encountered in cases of diabetes, but this occurs so infrequently that it is difficult to state whether or not the diabetic state has anything whatsoever to do with its occurrence.

Central Scotomas from Hereditary Degenerations: Leber's Disease and Hereditary Atrophy of the Optic Nerve.—Leber described a type of hereditary atrophy of the optic nerve with central scotomas in which males alone were affected and transmission was through females. Hereditary atrophy of the optic nerve, however, is not always sex limited. Many cases, likewise, have been reported in which central field defects were not discovered.

Thompson and Cashell [42] have described a pedigree in which 16 persons were affected throughout life in six generations. Ten persons

40. Landor, J. C., and Pallister, R. A.: Avitaminosis B_2, Tr. Roy. Soc. Trop. Med. & Hyg. **29**:121-134, 1935. Moore, D. G. F.: Retrobulbar Neuritis and Partial Optic Atrophy as Sequelae of Avitaminosis, Ann. Trop. Med. & Parasitol. **28**:295-303, 1934.

41. Box, C. R.: Amaurosis in Pernicious Anemia, Lancet **2**:1269, 1936.

41a. Kampmeir, R. G., and Jones, E.: Optic Atrophy in Pernicious Anemia, Am. J. M. Sc. **195**:633-638, 1938.

42. Thompson, A. H., and Cashell, G. T. W.: Pedigree of Congenital Optic Atrophy Embracing Sixteen Affected Cases in Six Generations, Proc. Roy. Soc. Med. **28**:1415-1425, 1935.

were males and 6 females. There were primary atrophy of the optic nerve and peripheral constriction of the visual fields rather than central scotomas, and inheritance was always through an affected mother. Nystagmus was either constant or intermittent in all affected persons. Griscom [43] published a somewhat similar pedigree involving three generations. Of 32 known members of the pedigree, 14 were affected, and of these, 8 were males and 6 females. The inheritance with one exception was through the father, who was affected. The patients exhibited paracentral scotomas and ring scotomas. The retinal vessels were narrowed, and there was some pigment disturbance in the periphery of the fundi. Probably these patients suffered from pigmentary degeneration of the retinas. Gunn [44] described atrophy of the optic nerve in a mother, in her son aged 4 and in her daughter aged 8 years. The mother had a central scotoma for red and green in her right visual field; otherwise her fields showed only concentric contraction. Alsberg [45] described primary atrophy of the optic nerve in a man aged 43, in whom symptoms commenced at 25, and in his son aged 7 years, who commenced to lose vision at the age of 4. In both patients vision was reduced to 20/200 or thereabouts. Herzog [46] described a grandmother, mother and son, all of whom had a primary type of atrophy of the optic nerve with resultant severe reduction in vision present during their entire lives. The mother's visual fields showed central scotomas, but those of the son showed concentric contraction. Hereditary atrophy of the optic nerve is usually bilateral, but some cases have been reported in which only one eye was affected.[47]

CASES 12 AND 13.—*Severe persistent reduction in visual acuity of both eyes of 2 sisters, aged 30 and 23, respectively; onset at 25 and 21 years, respectively; atrophy of the papillomacular bundle bilaterally with central scotomas; disseminated sclerosis seemed unlikely.*

E. P., a white woman aged 30, was seen in 1937. She complained of defective vision in both eyes. Her family history was interesting in that her sister had precisely similar trouble. After exposure to cold and a period of great anxiety due to a flood in the district in which she lived, she developed difficulty in reading. The visual loss was noticed first in the right eye and shortly thereafter in the left eye. For a period of several weeks at about this time there was complaint of pain on movement of the eye. The visual loss apparently occurred gradually.

At the time of examination some five years after the onset of the trouble vision in the right eye was 15/200 and in the left eye 5/200. There was bilateral

43. Griscom, J. M.: Hereditary Optic Atrophy, Am. J. Ophth. **4**:347-352, 1921.

44. Gunn, M.: Family Optic Atrophy in Mother and Two Children, Tr. Ophth. Soc. U. Kingdom **27**:221-225, 1907.

45. Alsberg: Hereditäre Sehnervenatrophie bei Vater und Sohn, Klin. Monatsbl. f. Augenh. **79**:832-833, 1927.

46. Herzog, M.: Vererbte, angeborene Sehnervenatrophie, Klin. Monatsbl. f. Augenh. **84**:536-543, 1930.

47. Handmann, in discussion on Alsberg.[45]

atrophy of the papillomacular bundle. Neurologic examination gave negative results. Special tests did not yield further information.

H.P., a white woman aged 23, complained of loss of vision over a period of two years. There was no complaint of pain or discomfort. Vision in both eyes was 1/200. There was bilateral atrophy of the papillomacular bundles.

The diagnosis of hereditary atrophy of the optic nerve is often loosely applied. There is little doubt that birth injuries are responsible for many so-called cases of hereditary atrophy of the optic nerve.

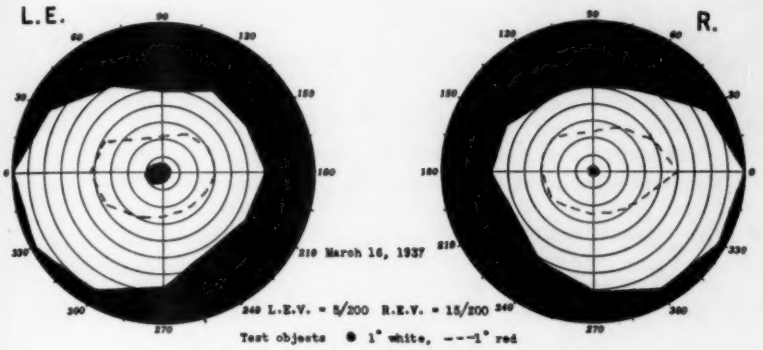

Fig. 10 (case 12).—Visual fields of E. P.

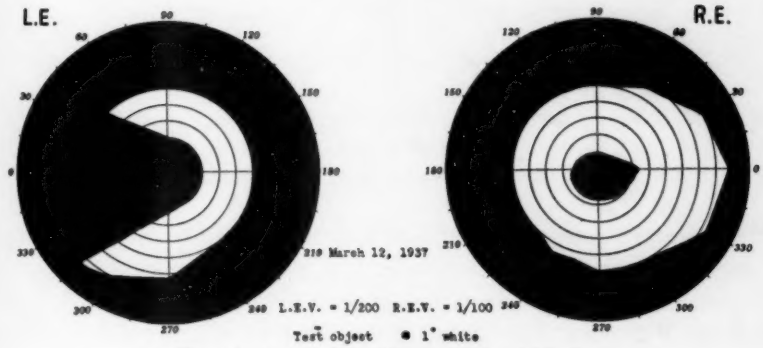

Fig. 11 (case 13).—Visual fields of H. P.

Through permission of Dr. G. Tayloe Gwathmey, of Orlando, Fla., we have had the opportunity of seeing a mother and son whose histories are reported here.

CASES 14 AND 15.—*Familial atrophy of the optic nerve in mother and son, both of whom had poor vision during their entire lifetime; atrophy of papillomacular bundle in both; cecocentral scotomas in mother's visual fields.*

H. B., a white woman of 41, complained of defective vision which had been present during her entire life. Her father had had a similar defect during his entire lifetime. She had four brothers, one of whom is reported to be similarly affected. She had always been healthy.

Examination of the eyes showed them to be normal externally. The nerve heads were clearly demarcated; normal physiologic cupping was present; there was pronounced atrophy of the papillomacular bundle. Vision in the right eye with a —6.00 sph. was 20/200; vision in the left eye with a —7.00 sph. was 20/300. The fields are presented in figure 12.

H. B., a white child of 3 years and 8 months, was noticed by his mother to hold things close to his face, and she guessed he might have inherited the defect from her. The child had always been healthy except for bilateral otitis media at 2 years of age. This had necessitated paracentesis of both ear drums.

External examination of the eyes showed them to be normal. Ophthalmoscopic examination revealed bilateral atrophy of the papillomacular bundle. Retinoscopic examination revealed an error in each eye of less than 2 diopters of hyperopia. The fields were normal for form on gross tests.

CONCLUSIONS

In conclusion, we should like to state that while this paper does not contain new material, it may serve to emphasize the importance of

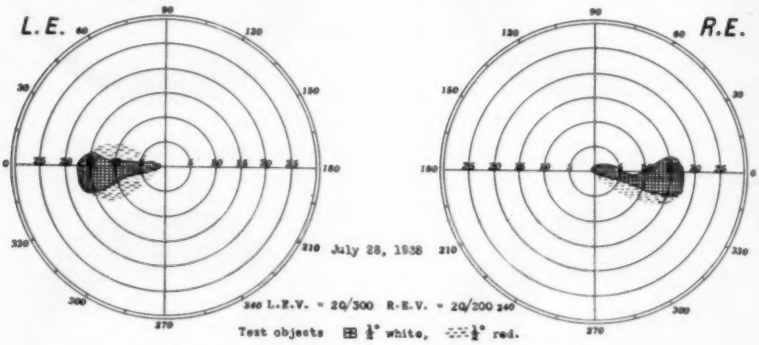

Fig. 12 (case 14).—Visual fields of H. B.

correct interpretation of central field defects. Attention has been drawn to the characteristics of these defects as accounted for by lesions at different levels: the macula, nerve, chiasm, tracts and radiation. Some conditions generally characterized by such defects have been discussed.

Dr. Dandy and Dr. Park gave us permission to use their material.

DISCUSSION

DR. JOHN EVANS: Reviews such as the one presented make for better physicians because they keep fresh those innumerable possibilities which form the basis for differential diagnosis.

I should like to stress for a moment certain points which have been discussed. The significance of the bridgelike connection between an enlarged blindspot and a central scotoma is often discussed, and the reference is often made to its fiber bundle origin. I cannot but feel that theories bearing on this type of defect are poorly supported by known anatomic and physiologic evidence. To elucidate this statement it is necessary to review some of the basic ideas referred to in this paper.

First, one must keep in mind the fact that the precipitating element is not the nerve fiber but the rod and cone and that when a central scotoma is being thought of in the purest sense of the word one is thinking of depressed cone function only. Supplementing this statement, I must stress the point that each foveal cone is said to be served by a single fiber, but a number of rods may be served by a single fiber. There is no reason to think that a single fiber might serve rods as well as cones, and this applies particularly to those conspicuously refined fibers which serve the closely packed cones of the fovea.

In regard to the relation of the papillomacular bundle to adjacent structures, this bundle occupies one third of the entire nerve head, sending its fibers directly to the macula. There is no reason to think that they serve rods or cones in the region of the retina between the papilla and the macula. The cecocentral region is really the nasal portion of the retina, because it is spared when the uncrossed or nasal fibers of the nerve are spared. The dividing line goes through the macula, not the nerve head. If one considers the path of the fibers serving this cecocentral region, it is seen at once that they pass into the nasal part of the nerve head. These fibers obviously arise from rods and from cones of the cecocentral region. Were they alone damaged there would be no central scotoma; only a paracentral scotoma would be expected, as the fibers of the cecocentral rods and cones would not have been affected.

It is equally obvious that destruction of the entire macular bundle would not account for such a bridge unless one postulates the existence of fibers in this specialized bundle, which also supply the cecocentral region. Even then, both crossed and uncrossed fibers of the papillomacular bundle would have to be mixed with crossed fibers which supply the rest of the cecocentral region, and this is so foreign to the meticulous separation of the crossed and uncrossed fibers in other parts of the nerve head that the idea seems hardly acceptable. As far as I know, experimental work tends to refute rather than support such an idea.

As a last consideration, fibers coming from the optic nerve immediately adjacent to the papillomacular bundle cannot form such a bridge of blindness, because they are uncrossed bundles and supply only rods or cones temporal to the hemianopic line. One would have to visualize a bandlike lesion passing horizontally through the nerve head and affecting uncrossed fibers of the nasal half and the papillomacular bundle located in the temporal part in order to produce the cecocentral bridge scotoma. There would probably be an associated defect in the nasal field. (I adhere to the Bund-Leber-Fuchs theory of fiber arrangement, which places fibers from peripheral portion of the retina in the axis of the nerve head.)

Perhaps one could work out some complex pattern which could explain these bridge defects, but there seems to be no evidence available to correlate this with the fiber bundle theory.

Basic evidence from another source on which a hypothesis could be built is not lacking. Certain known factors may be considered. The cones of the fovea derive their nourishment from the choroidal blood bed. The retinal system of blood vessels as seen ophthalmo-

scopically forms two invisible plexuses in the deeper layers of the retina. They can be traced to the region of the synapse between the bipolar cells and the rods and cones. Though the retinal system does not reach the foveal region, it is probable that the bipolar layer supplying its cones is reached by finer twigs, because the bipolar link lies almost parallel with the retinal surface instead of at right angles to it, as in other parts of the retina. There are two small vessels which reach out from the temporal border of the nerve head toward the macula. When they are large enough to be seen by the ophthalmoscope they are called cilioretinal vessels. It is apparently their function to supply the macular fibers, and they doubtless also send twigs laterally into the cecocentral zone. Derangements of this minute vascular tree doubtless cause interruptions in the passage of the nerve impulse from the cells in the region it supplies. When damage to the foveal region is central in origin or is due to damage purely of the fiber bundles, there may be a call for greater metabolic activity of this and other paramacular vessels as a gesture toward the restoration of local function. Such a mechanism is not unknown in other purely neural tissue. The lesion of anterior poliomyelitis in the spinal cord may be associated with circulatory disturbance in the affected extremities.

I think that more important from our point of view, however, is the fact that transient fluctuation of this "bridge" may be produced in many instances, thus demonstrating its circulatory basis. Widening of this bridge and of other parts of the angioscotoma often indicates the advent of increased intracranial pressure.

It is thus obvious that there is little evidence to sustain a neurogenic hypothesis to account for the cecocentral bridge, but there is obvious and conspicuous evidence supporting an angiogenic hypothesis.

OPHTHALMIA NODOSA

REPORT OF A CASE

F. N. KNAPP, M.D.
DULUTH, MINN.

Pagenstecher in 1883 described the first case of ophthalmia nodosa and termed the disease caterpillar hair ophthalmia. Wagenmann reported a case under the title pseudotuberculosis. Later Saemisch used the term ophthalmia nodosa.

There is an early initial stage soon after the caterpillar hairs have entered the eye in which the only symptom is the sensation of a foreign body in the eye. The patient complains of itching, burning, lacrimation and photophobia. All of these symptoms are aggravated by rubbing of the eyelids. There may be swelling of the lids, pericorneal injection and edema. On application of fluorescein, the cornea stains. The epithelium of the cornea is lost either by penetration of the caterpillar hairs or as a result of excessive rubbing of the eyelids. The discomfort is due to foreign body irritation and chemical irritation.

It has been demonstrated by Karsten that the canal of the hairs carries formic acid into the tissues. Gossen expressed the belief that the poison is a by-product of the organic material of the caterpillar hair which produces an external poison that is adherent to the hair. According to Stargardt's theory, the toxin is a by-product of the decomposition of the chitin brought about by absorption of the hair. De Schweinitz proved that the inflammatory reaction is not due to a secondary bacterial infection.

Some of the caterpillar hairs are not smooth but have fine barbs and spines which point toward the tip of the hair. The penetration of the hairs through the epithelium and their migration are accelerated by this barbed structure.

In the second stage of nodule formation microscopic study reveals a picture similar to that of a tubercle, with the exception that a hair is seen in the center of the nodule; hence the name pseudotuberculosis. On microscopic section the nodules consist of spindle cells and round cells, outside of which there is a capsular formation. Within the capsule are epithelioid cells and giant cells surrounding the hair.

There have been few cases of ophthalmia nodosa reported in the American literature. The first was reported by Knapp in 1897.[1] Col-

1. Knapp, G.: Ophthalmia Nodosa, Am. J. Ophth. **14**:247 (Aug.) 1897.

burn [2] reported another case during the discussion of Knapp's case. De Schweinitz and Shumway [3] reported a case in 1904. Parker [4] reported a case with an extensive review of the literature in 1910. Marlow [5] reported a case of ophthalmia nodosa in 1910. In 1924 there was a case reported by Grove.[6] All these were typical cases of ophthalmia nodosa, the symptoms appearing after injury by caterpillar hairs.

REPORT OF CASE

Mrs. W. R. S., aged 35, a housewife, was seen on June 30. About May 30 she complained of photophobia, lacrimation and itching in both eyes. In a few days there was rather severe pain. The pain was worse on rotation of the eyeballs. Later the pain radiated to the frontal and temporal regions. All of these symptoms continued to be present but were not so severe as at the onset. On repeated questioning she recalled transplanting flowers that were packed in hairy moss about two weeks before the onset of the ocular symptoms. The past history with reference to the eye was entirely normal. Vision in the right eye was 20/20 and in the left eye 20/20. The skin of both upper and lower eyelids was slightly edematous, and there was a little hyperemia of the palpebral conjunctiva. On eversion of the upper lid of each eye the bulbar conjunctiva presented many small yellow nodules from the fornices to within 3 mm. of the limbus. Some of the nodules were round; others were elongated, and all were situated beneath the conjunctiva. There was no staining of the conjunctiva or cornea by fluorescein. No discharge was present. The conjunctival blood vessels were engorged in the area of the nodules. The lower half of the bulbar conjunctiva was slightly edematous, but no redness was present.

A solution of mercuric oxycyanide (1:1,000) was repeatedly injected in the vicinity of the nodules. Only enough to make a small bleb was used in these treatments. The injections were repeated eight or nine times at intervals of three days. After injection of mercuric oxycyanide the nodules rapidly disappeared in the area in which the injection was made.

Sections were submitted to several physicians for biopsy (figs. 2 and 3). Dr. A. Berdez reported: "The sections show a tissue of granulation with several nodules measuring up to 0.03 by 0.15 cm.; the nodules are formed by a tissue of granulation showing numerous giant cells of the foreign body type, lymphocytes and proliferated fibroblasts. The central part of the nodules is formed by a reddish amorphous material in which faintly bluish-staining formations can be seen which suggest broken-up mycelium. Sections stained with Ziehl's solution for acid-

2. Colburn, J. E., in discussion on Knapp.[1]

3. de Schweinitz, G. E., and Shumway, E. A.: Conjunctivitis Nodosa with Histologic Examination, Univ. Pennsylvania M. Bull. **17**:270 (Nov.) 1904.

4. Parker, W. R.: Ophthalmia Nodosa or Caterpillar-Hair Ophthalmia, J. A. M. A. **55**:639 (Aug. 20) 1910.

5. Marlow, F. W.: Report of a Case of Ophthalmia Nodosa, Arch. Ophth. **39**:107 (April) 1910.

6. Grove, B. H.: Caterpillar Conjunctivitis, Am. J. Ophth. **7**:140 (Feb.) 1924.

Fig. 1.—Photograph of patient showing nodules in the upper fornix of the right eye.

fast organisms show nothing of note. The lesion is not tuberculosis, tularemia or ordinary foreign body reaction; the lesion represents a granulomatous inflammation of some other etiology, apparently due to a fungus. Cultures made on Sabouraud's agar and on blood agar have remained sterile. Diagnosis: Tissue of granulation from the conjunctiva (fungus infection)."

Dr. Arthur T. Henrici submitted the following report: "The peculiar giant cell reaction is characteristic of fungus lesions but could also result from a foreign

Fig. 2.—Low power photomicrograph showing multiple granulomas.

body reaction. There are in the sections some basic staining, fine fibrous structures which look somewhat like mold mycelium which is distorted or disintegrating, but these structures are not gram-positive, as they should be if they were truly mycelium. Similar fibrillar structures may sometimes develop from compressed tissue elements in an inflammatory reaction.

"In short, if this is a fungus infection, the fungi have been so far destroyed by the inflammatory reaction as to become gram-negative and not definitely recognizable. It could be a simple foreign body reaction."

Dr. F. H. Verhoeff reported: "I feel sure that your case is one of ophthalmia nodosa. Classically this is caused by plant hairs. I cannot recognize any cater-

pillar hairs in the foreign body tubercles, nor definite plant hairs, but often this is so in such cases. However, I do find a few structures that could be plant hairs and places in which there may be disintegrating plant hairs. If no recurrence takes place the condition in all probability was due to plant hairs, for when due to caterpillar hairs new tubercles continue to develop for a long period of time due to migration of the hairs. The fact that there is very slight inflammatory reaction around the tubercles seems to exclude fungus infection. The apparent

Fig. 3.—High power photomicrograph showing giant cells in a granuloma.

filaments in the centers of some of the tubercles probably have resulted from the cell reactions."

The patient in this case first presented irritation after exposure to hairy moss. Later nodules appeared beneath the conjunctiva. As the lesions did not recur, as is characteristic of ophthalmia nodosa from caterpillar hair, it seems logical to agree with Dr. Verhoeff that this case is one of ophthalmia nodosa in which plant hairs were the exciting agent.

TRANSCRANIAL EXTIRPATION OF A FIBRO-HEMANGIOMA OF THE ORBIT

REPORT OF A CASE

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Fellow in Ophthalmology
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Hemangioma of the orbit is considered by Benedict and Love,[1] Reese [2] and others to be the most common type of primary intraorbital tumor. The typical case is one in which slowly developing but variable unilateral exophthalmos occurs, usually in the first or second decade of life. Exophthalmos is usually "straight forward," and the degree of exophthalmos may increase with dependent posture of the head or with compression of the jugular vein. It is not usually pulsatile, nor is a bruit commonly heard. Vision is not significantly affected unless the tumor becomes large or is situated within the muscle cone. Limitation of ocular rotations seldom occurs. A portion of the cavernous type of angioma is sometimes present in the lid or conjunctival sac, but the fibrous type, which is usually located in the posterior portion of the orbit, may give no external evidence of its nature.

Unilateral exophthalmos is frequently the only symptom presented by the patient with a vascular tumor of the orbit. Its presence indicates the need for exhaustive study to exclude exophthalmos of extraorbital origin. If one suspects a primary tumor to exist in the orbit, hemangioma should be among the first possibilities to consider in the differential diagnosis. Rapid regression of exophthalmos after irradiation may confirm the diagnosis, since this type of tumor is quite radiosensitive. It must be admitted, however, that a careful study of many cases of unilateral exophthalmos frequently fails to establish a preoperative pathologic diagnosis or even an exact anatomic location of the lesion.

From the Department of Ophthalmology, Hospital of the University of Pennsylvania.

Read before Section on Ophthalmology, College of Physicians of Philadelphia, Dec. 21, 1939.

1. Benedict, W. L., and Love, J. G.: Cavernous Hemangioma of the Orbit with Hyperostosis, Am. J. Ophth. **22**:1149, 1939.

2. Reese, A. B.: Symposium: Etiology of Exophthalmos; Complications; Causes from Primary Lesions in the Orbit; Surgery, Tr. Am. Acad. Ophth. **39**:65, 1934.

The uncertainty of the position and nature of the growth makes the choice of operation difficult if operation is decided on. The orbital approach, as outlined by Elschnig,[3] may be directed through the conjunctiva, through the skin of the lids or through an opening created by an osteoplastic flap of the malar bone—the Krönlein procedure. Exenteration of the orbit, of course, constitutes another operative measure. The usual approaches are satisfactory for tumors in the anterior aspect of the orbit. They frequently prove inadequate, however, when removal of a tumor from the posterior portion of the orbit is attempted. Unnecessary trauma to vital orbital structures may result, or the tumor may be incompletely removed, a fact of serious importance if intracranial extension has taken place. It must also be remembered that copious hemorrhage, which occurs commonly in the removal of highly vascularized tumors, may be extremely difficult to control.

The inadequacy of the usual methods of approach to the orbit has led to a consideration of a transcranial procedure. Benedict and Adson[4] in 1934 reported a case in which transfrontal craniotomy was done with unroofing of the orbit to effect the removal of an intraorbital tumor. Naffziger[5] previously hinted at the feasibility of the operation in his description of orbital decompression for malignant exophthalmos. Dandy[6] in 1922 reported a somewhat similar procedure for the treatment of intracranial tumors of the optic nerve. The approach is obviously neurosurgical, but it does not possess the gravity of the usual craniotomy, since it is entirely extradural. The procedure permits satisfactory exploration of the orbit and contiguous cranial cavity and offers accessibility to tumors in these areas.

The case reported here represents one in which a retrobulbar orbital neoplasm was suspected. A transcranial surgical approach to the orbit was made which permitted successful, complete removal of an encapsulated fibrohemangioma of the posterior orbit.

REPORT OF CASE

F. C., a 34 year old white man, was admitted to the service of Dr. Francis Heed Adler at the Hospital of the University of Pennsylvania on Aug. 29, 1939 for the treatment of a protruding left eye. The condition was first noticed six

3. Elschnig, A.: Operations for Tumor of the Orbit, Surg., Gynec. & Obst. **45**:65, 1927.

4. Benedict, W. L., and Adson, A. W.: Hemangio-Endothelioma of the Orbit: Removal Through Transcranial Approach, Tr. Sect. Ophth., A. M. A., 1934, p. 79.

5. Naffziger, H. C.: Progressive Exophthalmos Following Thyroidectomy: Its Pathology and Treatment, Ann. Surg. **94**:582, 1931.

6. Dandy, W. E.: Prechiasmal Intracranial Tumors of the Optic Nerves, Am. J. Ophth. **5**:169, 1922.

years previously, at which time the patient was told that his left eye was more prominent than the right. No precipitating event was known to have caused the exophthalmos, but the patient recalled that he sustained a moderate contusion in his left temporal region with a blunt metal object in 1930. No additional ocular difficulties were observed until 1937, at which time the patient complained of blurring of vision and vertigo. These symptoms were alleviated with a suitable myopic correction. The patient later experienced transient knifelike pains in the left frontal region which radiated to the temple.

Exophthalmos of the left eye was variable during the six years in which it was observed. Occasionally it was of such degree that the lids could not be closed. In the period immediately before admission the patient experienced several instances during which the eye prolapsed from the orbit, but it was reduced without much difficulty.

The patient's past medical and family histories were essentially unimportant. Systemic review disclosed no relevant facts.

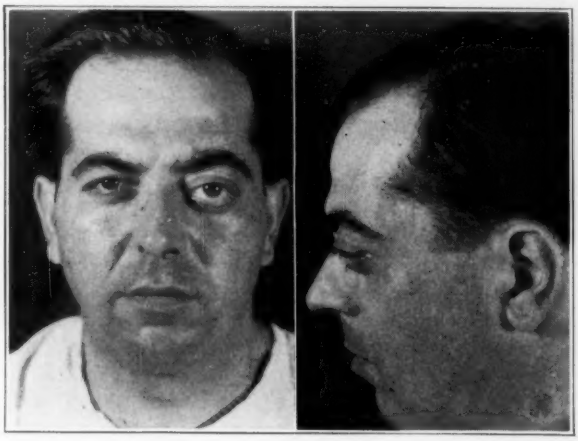

Fig. 1.—Unilateral exophthalmos produced by a fibrohemangioma of the left orbit.

Physical examination revealed a large, well developed young white man with an obvious exophthalmos on the left side. No other remarkable abnormality was observed on physical examination. The results of ocular examination were as follows: Vision with correction was 6/9 in the right eye and 6/12 in the left. The exophthalmometer (Hertel) reading for the right eye was 19 mm. and for the left 27 mm. Intraocular tension was 15 mm. of mercury (new Schiötz) in both eyes. The left palpebral fissure was wider than the right because of a proptosis of the left eye, which was essentially "straight forward." The conjunctiva and cornea were normal in each eye. The pupils were round and equal and showed normal reactions. Ocular rotations of the left eye were slightly limited in the left upper quadrant of gaze, but no diplopia was elicited. There was a suggestion of a soft mass palpable toward the outer aspect of the left upper lid. Ophthalmoscopic examination of the right eye disclosed a normal fundus with moderately tortuous veins. The left eye presented a disk with a moderately blurred nasal margin. The retinal veins in this eye were somewhat distended when compared

with those of the right eye. Two small patches of healed choroiditis were present in the temporal periphery of the retina.

Visual fields of the right eye were normal. Study of the central visual field of the left eye showed a marked increase in the size of the blindspot, especially to a 1 mm. test object at a distance of 1 meter. The field for red was also markedly diminished in this eye. A diplopia field failed to disclose diplopia in the cardinal directions of gaze.

A blood count was normal. The level of the blood cholesterol was moderately elevated (to 278 mg. per hundred cubic centimeters). A basal metabolic rate was —7 per cent. Kahn and Wassermann reactions of the blood were negative. Lumbar puncture revealed cerebrospinal fluid under normal pressure. The cell count and protein content were normal. A weakly positive Wassermann reaction of the spinal fluid was reported, but this finding was refuted on two subsequent occasions. The colloidal gold curve of the spinal fluid was insignificant. The patient showed no cutaneous reaction to significant quantities of tuberculoprotein (purified protein derivative).

Roentgen studies revealed no abnormalities of the skull, orbits or sinuses.

At the conclusion of clinical examination it seemed probable that the exophthalmos was due to a benign tumor of the posterior portion of the left orbit. In view of the fact that the nature of the tumor could not be accurately decided on and because of its apparent posterior location, a transfrontal approach to the orbit was favored for its removal. The patient was consequently transferred to the neurosurgical service.

On September 15 Dr. Francis C. Grant turned down a small frontal bone flap of the type used for approach to the pituitary gland. Since no tumor was discovered in or about the sella turcica, the orbital roof, which had been exposed extradurally, was then removed. In rongeuring away the orbital roof Dr. Grant was careful not to carry the opening too far mesially, to avoid the hazard of penetrating the ethmoid sinuses. An incision was made in the periorbita, and herniation of orbital fat into the incision followed. This tissue was dissected away by Dr. F. H. Adler until the muscle cone was reached. A mass was seen to occupy the posterolateral aspect of the orbit; it was dark blue and apparently well circumscribed. After a little blunt dissection the tumor was completely enucleated without rupturing its capsule. There was no bleeding after its removal. The periorbita was approximated with sutures, after which the frontal bone flap was replaced and wired. The incision in the skin was closed with interrupted sutures.

On the day after operation there were a good deal of edema and ecchymosis of the lids, the usual accompaniment of this type of craniotomy. The patient's general postoperative state was excellent. He was ambulatory on the sixth day. The skin sutures were removed at this time, and three days later the patient's condition was satisfactory enough to permit his discharge from neurosurgical care. Measurement with the exophthalmometer on the ninth postoperative day was 19 mm. for the right eye and 22.5 mm. for the left. Ecchymosis and edema of the lids began to subside during this period, but a moderate amount of blepharoptosis, apparently mechanical, was seen. Ophthalmoscopic examination of the left eye revealed the persistence of blurring of the nasal margin of the disk, but the retinal veins were reduced to normal in caliber. Another study of the central visual field of the left eye showed considerable improvement, although the blindspot remained somewhat enlarged. Uncorrected visual acuity of the left eye was 6/12. There was no visible limitation of ocular rotations.

Pathologic study of the tumor was made by Dr. F. H. Lewy (fig. 2). Many thin-walled blood vessels interspersed with precollagenous fibers and occasional fibroblasts were observed. The diagnosis of fibrohemangioma was suggested.

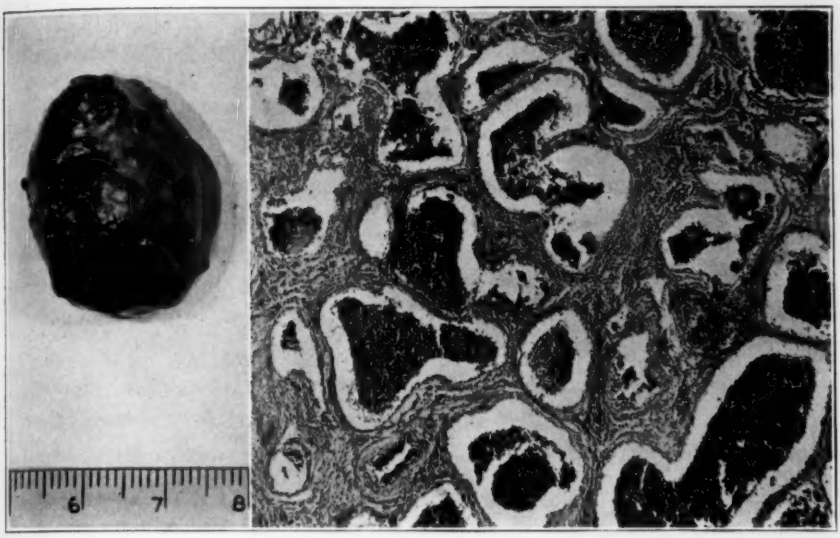

Fig. 2.—Gross and microscopic (× 37) appearance of the tumor.

Fig. 3.—Regression of exophthalmos two weeks after transcranial removal of fibrohemangioma.

The patient was discharged from the hospital on September 29. He was seen again on October 20, at which time there was no measurable exophthalmos of the left eye and only a slight residuum of ptosis. The craniotomy incision was entirely healed.

A SIMPLIFIED OPERATION FOR KERATOPLASTY

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AND
M. I. GREEN, M.D.
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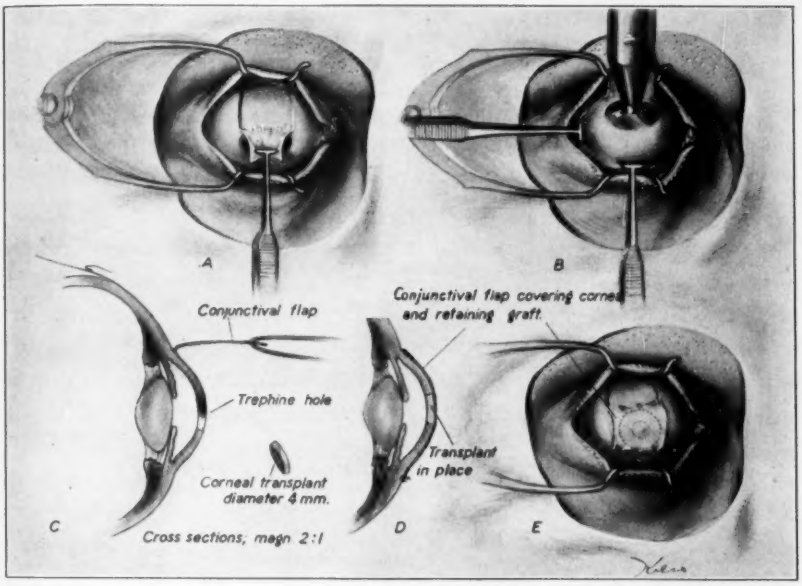
The transplantation of clear corneal tissue from an enucleated eye to an opaque eye has never developed any real popularity in the past, mainly because so few results were successful. There were two principal reasons for the failure: first, and most important, the difficulty of obtaining healthy, clear corneas, and second, the complicated technic.

These difficulties have been largely solved by the epoch-making contributions of Filatov. He discovered that a section of cornea taken from a healthy patient who had just died as the result of an accident could be used successfully for transplantation in a favorable eye. He later found that by keeping the eye under aseptic conditions for two or three days at a temperature of about 4 C. the results were even better than when the eye was used immediately after enucleation. This solved the problem of obtaining sufficient desirable material whenever needed. He also simplified the method of keeping the graft in place without putting sutures into the corneal tissue itself by using a flap of the full width of the upper conjunctiva, everting it so that only the epithelial surface touched the corneal wound and retaining it by suturing it to the lower conjunctiva.

Since our visit to Professor Filatov in Odessa, U. S. S. R., we have devoted a great deal of study to experimentation in order to simplify the operation of corneal transplantation so that any one with average skill might be able to perform it. We have experimented with various types of trephines, a revolving keratome and a punch. We have come to the conclusion that an automatic trephine with a 4 or 4.5 mm. blade guarded with a shoulder extending 1 to 1.5 mm. from the cutting edge is the most efficient and easiest means of obtaining a corneal disk. Although one blade could be used, two blades would be far more convenient. They may both be of the same circumference, but a slight difference in length will often be found necessary to remove the opaque disk from the recipient's eye, as it will usually be found thicker than the normal cornea, owing to the pathologic condition which caused the opacity.

TECHNIC

The recipient's eye is prepared as for a cataract operation, with complete akinesia. One cubic centimeter of a 4 per cent solution of procaine hydrochloride and epinephrine hydrochloride is injected retrobulbarly. The lids are anesthetized by the O'Brien method and local injections. The conjunctiva and cornea are anesthetized by using 4 drops of a 4 or 5 per cent solution of cocaine hydrochloride every five minutes. A horizontal incision is made in the conjunctiva in the upper fornix, about 12 mm. wide, and the conjunctiva is dissected down to the limbus. Three black silk sutures are introduced in the distal end of the flap, and the eye is closed.



Technic of operation. *A* shows the conjunctival flap; *B*, the trephine incision to remove the disk of cornea from the recipient's eye; *C*, cross section of the eye previous to receiving the corneal transplant; *D*, the transplant in place; *E*, the conjunctival flap sutured over the transplant.

The next step is to obtain a disk of clear cornea from the donating eye, which has been kept in a refrigerator at 4 C. for three days. Repetition of the method of keeping the eye may be convenient: The eye should be removed from the cadaver within twenty-four hours after death. Before removal, the cul-de-sac is flushed with a 1 per cent aqueous solution of brilliant green or a 1:5,000 solution of mercuric cyanide. The eye is enucleated under aseptic conditions and placed in a sterile specimen jar without solution of any kind, there being enough moisture in the ocular tissue to keep the eye moist for the next three days. The jar is covered with its metal lid.

The 4 mm. blade with the 1 mm. shoulder is mounted on the automatic trephine, and the disk is cut out cleanly from the donating eye and left in the blade. If the disk happens to drop out of the blade, it must be placed on a moist watch

glass or a strip of rubber tissue, care being taken to note whether the anterior or the posterior surface is up.

The longer blade, with the 1.5 mm. shoulder, is next mounted on an automatic trephine, and the disk of opaque cornea is cut out of the recipient's eye. Care is taken to place the clear disk with the epithelial surface upward in the recipient cornea, the clear disk being smoothed out so that it does not project above the recipient cornea. A drop of a 2 per cent solution of atropine is placed in the cul-de-sac. The prepared conjunctiva is turned down over the cornea, the unbroken epithelium of the conjunctiva in contact with the transplanted cornea, and pulled down carefully and sutured in place to the conjunctiva at the limbus below. If desired, an additional suture may be placed in the everted conjunctiva at the side and stitched to the adjoining conjunctiva at the limbus. Some ointment containing atropine is placed in the cul-de-sac, and the eye is closed as for a cataract operation, with a pad and strips of adhesive tape.

The eye must not be investigated for one week. Failure to observe this rule is responsible for most failures, as many ophthalmologists are sufficiently curious to dress the eye one, two or three days after the operation. This is the reason why so many ophthalmologists use corneal sutures to keep the disk in place. These will not be necessary if that curiosity can be suppressed and the operation is performed as indicated.

AN OPERATION FOR SPASTIC ENTROPION

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The treatment of spastic entropion has in my experience not been entirely satisfactory, and in talking with other ophthalmologists I find that they have had similar disappointments. This applies to the treatment of entropion which has not responded to injections of alcohol, the use of sutures or other therapeutic measures.

Some of the numerous procedures for the relief of entropion follow, with a description of the principles involved in a number of them:

(*a*) Temporary mechanical correction by means of collodion, adhesive plaster, sutures or cautery puncture.

(*b*) Correction by plastic procedures, such as the Celsus operation, Graefe's operation, Vogt's canthoplasty and the Panas-Chronis procedure. These procedures evert the lid by shortening the skin from above down, as in the Celsus operation, or by making the skin taut from side to side, pushing backward on the orbicularis oculi muscle and the lower border of the tarsus, thereby correcting the faulty position of the cilia. The skin is a poor antagonist to the muscles and soon stretches.

(*c*) Procedures to induce paralysis of the muscle or group of muscles causing the entropion or to weaken their action, such as the injection of alcohol as practiced by Wendell Hughes, or extirpation of Riolan's muscle or complete excision of the palpebral fibers of the orbicularis oculi muscle. Weakening one group of muscles may cause the other to act more forcibly, and complete removal of the orbicularis oculi muscles may produce ectropion, as no muscles are left to hold the lid against the eye.

(*d*) Wheeler's method for strengthening the action of the palpebral fibers of the orbicularis oculi muscle. This effect is accomplished by overlapping or by stretching the palpebral fibers upward and outward to a new attachment. The procedure usually intensifies the action of these fibers. This new attachment may relieve the entropion at first, but later the fibers at the margin of the lid may cause inversion of the cilia by spasm of the marginal fibers.

(*e*) Tarsectomy, as practiced by Kuhnt, to relieve a deformed position of the tarsus. Of course if the muscle forces causing the entropion are not corrected, the lid may continue to turn in.

Read before the New York Academy of Medicine, Jan. 15, 1940.

(*f*) Removal of the cilia, as in Flahrer's ablation of the cilia.

For relief of entropion in the early stage, as after the removal of a bandage, sutures, injection of alcohol or the application of adhesive plaster should be tried. If entropion does not respond to these measures, some new method needs to be employed.

For a long time I have felt that the procedures adopted were not applicable to the anatomic conditions which bring about entropion. In looking through Berens'[1] "textbook of ophthalmology" some months ago I found that McCool[2] expressed exactly the same idea regarding the anatomic conditions that I have found present in the lower lid of the eye. He stated that the palpebral fibers of the orbicularis oculi muscle form arcs, the concavity of which is pointed in two different directions. One type of curvature surrounds the palpebral fissure and directs its concavity toward the fissure; the other type of curvature is caused by the fibers being molded to the eyeball with the tarsus. The concavity of this type is directed toward the eyeball. With deficient support of the free border of the eyelid and an excess of skin on the lower lid, contraction of the palpebral fibers of the orbicularis oculi muscle takes place, and what was formerly an arc becomes the chord of an arc. The fibers exert a double action, narrowing the palpebral fissure and pressing the tarsus against the eyeball. Either action may cause entropion.

This pressure against the eyeball is the more important because the lower lid is in perfect contact with the eyeball only so long as the eyeball gives proper support. When there is enophthalmos, an empty socket, a small eyeball or lack of orbital fat, as is seen in senile persons with sunken eyes, the free edge of the lid receives insufficient support posteriorly and the tarsus may bend in or out, giving rise to entropion or ectropion. The character of the skin is important because a large amount of loose wrinkled and readily displacable skin contributes to the formation of the entropion. A healthy, firm skin with good tone will not readily give way.

Blepharospasm from irritation of the cornea or from pressure of a bandage worn too long pulls the skin over the free border of the lower lid, giving the necessary impetus to inversion of the lid. Busacca stated that in cases of entropion the orbicularis oculi muscle is often hypertrophied.

The following operation has been devised with the direction of force of the muscle in mind, in order to attempt to relieve the entropion permanently.

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1. Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936.

2. McCool, J. L.: Diseases of the Eyelids, in Berens,[1] pp. 360-361.

TECHNIC

After the skin of the eye is carefully washed with soap and water, it is dried. A 3 per cent solution of iodine is applied, and the excess is removed with a swab dipped in alcohol. The lashes are carefully painted with iodine, and the conjunctival sac is flushed with warm boric acid solution. A 2 per cent solution of procaine hydrochloride, with 1 drop of epinephrine hydrochloride to the cubic centimeter, is injected subcutaneously along the entire length of the lower

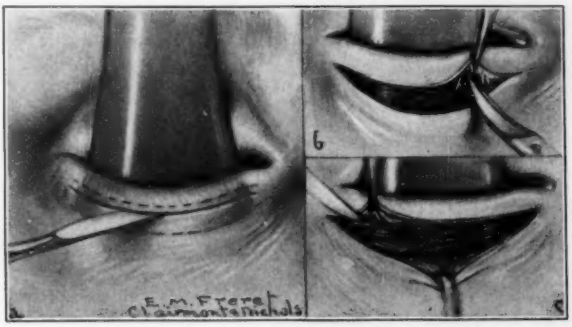


Fig. 1.—In *a* an incision is made 3 mm. below the margin of the lid and the entire length of the lid. The broken line represents the area of skin to be undermined. In *b*, the upper skin flap is undermined 1 mm. and the lower skin flap 3 mm. In *c*, the muscle flaps are outlined with a knife.

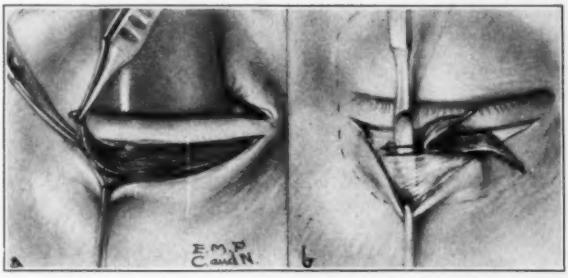


Fig. 2.—In *a*, a muscle flap is dissected up at the temporal side. In *b*, the two muscle flaps are dissected up, leaving 7 mm. of muscle still attached with its center slightly temporal to the center of the lower margin of the lid. A tunnel is made with a knife down to the anterior surface of the inferior orbital margin, 25 degrees from the center of attached muscle on the temporal side. A similar tunnel is made on the nasal side.

lid from the margin, covering an area 1 cm. wide. The under, concave surface of the broad end of a Jaeger plate is greased with petrolatum and slipped under the lower lid. The concavity is toward the eyeball.

An incision is made the entire length of the lower lid 3 mm. from the margin. The lower flap is undermined 3 mm., and the upper one is undermined 1 mm. Two flaps of orbicularis oculi muscle, each 4 mm. in width and tapering at each

end, are first outlined with a knife and then dissected up from the tarsus, one from the direction of the external canthus and one from the direction of the internal canthus. The muscle is left attached to the tarsus in an area 7 mm. wide with its center just temporal to the center of the lower margin of the lid. An incision is made with a knife, 25 degrees temporal from a point below the center of this attached muscle down to the anterior surface of the inferior orbital margin.

A suture of 000 plain catgut is passed through the periosteum. A Yankower needle is useful here. The muscle flap is loosely sutured into place on the

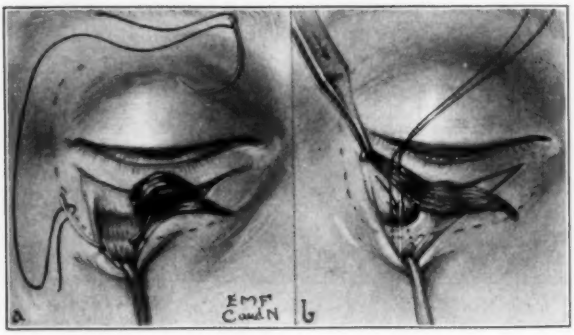


Fig. 3.—In *a* a suture is placed in the periosteum of the inferior orbital margin on its anterior surface. In *b* the suture is passed through the temporal muscle flap, and then the nasal muscle flap is similarly treated.

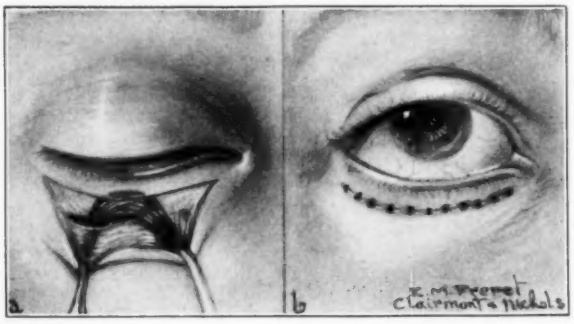


Fig. 4.—In *a* the temporal flap is sutured loosely into place. The nasal flap is similarly sutured loosely to the anterior surface of the inferior orbital surface, 25 degrees nasal to the center of the attached orbicularis oculi muscle. In *b*, the skin wound is closed with interrupted black silk sutures.

anterior surface of the inferior orbital margin. A similar incision is made 25 degrees nasal to the center of the attached muscle, and the other flap is sutured loosely in place on the anterior surface of the nasal orbital margin. The skin is then closed with interrupted black silk sutures. A pressure dressing is applied, and the wound is not dressed for three days. If the skin sutures are fine black

silk tied firmly at the very edge of the skin, many will come away with the dressing. Where there is an overabundance of skin, it may be excised according to the Celsus procedure. In place of catgut, a double-armed no. 1 black silk suture may be used; it is drawn through the periosteum and then the muscle and comes out through the skin, first through the temporal flap and then through the nasal flap. These sutures are tied over rubber pegs. They are removed on the fifth day.

Infection may be prevented by careful antiseptic and aseptic precautions. Sterile rubber gloves should be worn.

The use of epinephrine hydrochloride will prevent excess bleeding. The pressure bandage usually stops subsequent bleeding.

Brawny infiltration due to cutting the lymphatics may occur. There is no way I know to prevent this.

The flaps of muscle must be attached loosely to the anterior surface of the orbital rim, because it is easy to overcorrect the defect and draw the lid away from the eye if the muscles are tightly attached.

This operation, I believe, fulfils Spaeth's three requirements for a successful operation for entropion: It relieves the faulty position of the cilia. It prevents recurrence. It corrects the defect with the least amount of disfigurement.

DISCUSSION

DR. WILLIS S. KNIGHTON: I should like to ask Dr. Meek if this type of operation can be applied in cases of entropion in which the lower lid sags and turns in at the same time. I believe that one of the objectives of the operation Dr. Wheeler devised was the sling effect of the operation, which lifts up the lower lid and also corrects the inversion.

DR. SIGMUND A. AGATSTON: Dr. Meek speaks of the muscular action in the vertical direction. I believe that it is probable that there is a mechanical action in his operation. I wonder whether he severed the nerve supply to that muscle in doing his operation.

DR. RAYMOND MEEK: In reply to Dr. Knighton, I believe that this operation could be done when there is sagging of the lower lid, but the primary object is to correct the entropion. Later one might have to do a resection of the external canthal ligament in order to pull the lid up into place.

In regard to Dr. Agatston's question about severing the nerve supply, I believe that the nerve supply to a great part of the orbicularis oculi muscle should be severed, but there is still the portion above the incised muscle which is not touched and the subtarsal portion of the orbicularis oculi muscle, back of the tarsus, both in the lower lid. These muscles would still have their innervation.

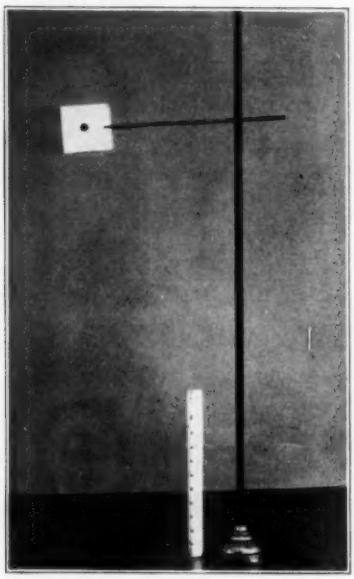
Clinical Notes

CORNEAL MOLDING FOR CONTACT GLASSES

Adjustable Fixation Target and Simplified Procedure for Removing Mold

JULIAN F. CHISHOLM JR., M.D., BOSTON

One of the greatest difficulties in satisfactory molding of contact glasses has been in securing adequate fixation. Obrig [1] has suggested that the patient's own glass be held over the other eye when the vision is



Adjustable fixation target.

impaired. This helps in fixation. However, when the vision is considerably reduced or the patient is amblyopic in one eye, it is of no real value.

To overcome this, an adjustable arm for a marker was devised, as shown in the accompanying illustration. The base of a student's lamp was used. A pipe about 5 feet (152 cm.) long was threaded to fit it. Holes were drilled through the pipe approximately every 6 inches (15 cm.). Into these fits a small rod about 12 inches (30 cm.) long, which carries a card. The carrier can be adjusted in and out by sliding it through the hole. A heavy black circle containing a large black dot in

From the Harvard Medical School and the Massachusetts Eye and Ear Infirmary.

1. Obrig, T. E.: Fitting of Contact Lenses for Persons with Ametropia: Evolution and Modern Technic, Arch. Ophth. **17**:1089 (June) 1937.

the center of the card serves as the marker. The distance of the marker from the patient depends, of course, on the amount of vision. There is no need to adhere to this type. The apparatus is rather simple to make and not cumbersome to handle. The idea more than the model is important.

Given a one-eyed patient or one with an amblyopic eye, the marker can be lowered so that the patient may grasp the marker carrier in his fingers and fix on them. The marker is then so set that the patient is looking in the direction that the physician desires, usually a little superiorly and slightly nasally.

After the mold is made, satisfactory removal of it is often difficult. To the patient this is the most unpleasant part of the entire procedure. To a large extent this discomfort can be overcome by having the patient rotate his eyes upward as far as possible. A muscle hook is applied to the sclera at the lower edge of the mold and steady pressure exerted. After a little dimpling of the globe is produced, air enters between the mold and the globe. If at the same time the handle of the molding shell is grasped in the other hand, the shell can either be felt to separate from the globe or at times is seen to ride out from between the lids by itself. The patient usually experiences only a moderate amount of discomfort from the pressure and no real pain.

BIFOCAL LENSES

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The report of a cosmetically improved myodisk lens by Dr. Andrew Hunter in the April issue of the ARCHIVES, page 839, is especially interesting because it illustrates that those supposedly best informed on the making of lenses are not necessarily always right. A little independent thinking and observation on the part of the physician who comes in actual contact with the patient may disprove some standardized notions of even the leading manufacturers. A case in point refers to bifocal lenses. At a recent ophthalmologic meeting the technical expert of a leading manufacturer of lenses said that the ideal bifocal lens is one having two optical centers, about 8 mm. apart. A bifocal lens with a single optical center, he said, in substance, causes prism effects which are confusing to the patient. The patient is apt to miss steps in going up or down because the step is not where he sees it; it seems higher or lower because of the prism effect at the reading portion. The indistinctness of the step seen through the reading portion is not disturbing, but the displacement is. To eliminate the prism effect, he said that the ideal bifocal lens is one with two optical centers.

Now this "ideal" lens may well be questioned. It seems more likely that it is just the opposite, namely, a bifocal lens with a single optical center both for distance and for reading. To make this plainer, the optical center of the distance and reading portion should coincide. Then the incident light from an object, striking any portion of the bifocal lens, will have its image formed along one principal or secondary axis. The first bifocal lens, the Franklin split bifocal, was a monocentric lens;

that is, the optical centers of the upper and lower half-lenses coincided. A later modification of it, the perfection bifocal lens, also was monocentric, and except for appearance is an ideal bifocal lens. Franklin, according to his report was happy with his bifocal lenses and experienced none of the difficulties that present day patients do. Possibly if Franklin had started with a modern bicentric "jump" bifocal lens, he would have given up the idea of the feasibility of bifocal lenses as some patients do today.

The difficulties patients have with modern bifocal lenses is probably largely due to the lenses having two separated optical centers, that is, being bicentric. This causes an image to be formed along two axes. If both sets of light pencils enter the pupil, the object is seen double. If the pupil happens to lie between the two sets of light pencils, neither one enters the eye and there is a gap or blind area. The so-called "jump" experienced when the eye changes from the upper to the lower portion is most likely due to the doubling and the gap. Similarly, the patient is apt to miss a step and misjudge the position of objects because of the doubling effect and the blind area defects which are inherent in all bicentric bifocal lenses.

The prism value at the reading portion is of little or no significance as long as there is no appreciable difference between the lenses for the two eyes. For example, one can easily get about with, say, a 4 prism diopter, base down or up, for each eye without missing steps or having to fumble for an object. If the step is raised or lowered, so is the foot raised or lowered and to the same extent, so they make perfect contact. The same applies for things to be grasped by the hand. The prisms displace the object and the hand to the same extent, so there is no fumbling. The manufacturers have produced almost ideally invisible bifocal lenses from the cosmetic point of view, but optically it seems they may be on the wrong track if their ideal is a bicentric bifocal lens.

Ophthalmologic Reviews

PHARMACODYNAMICS OF THE INTRAOCULAR MUSCLES

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Effects of the commonly used "miotics" and "mydriatics" on the size of the pupil and the state of accommodation are generally well known to the ophthalmologist. However, records of the quantitative effects of these drugs are often difficult to find and are sometimes contradictory, and even the qualitative effects are in certain instances either controversial or not widely known. The actions of these drugs are reviewed from a physiologic standpoint, with a summary of the quantitative data whenever possible.

An understanding of the reactions of the autonomic nervous system in general, especially with regard to the concept of chemical mediation of nerve impulses, is essential to an interpretation of the effects of various drugs on the intraocular muscles. In this review the subject is approached by (1) summarizing the concept of chemical mediation as related to the actions of various groups of drugs on the autonomic nervous system as a whole, (2) reviewing the reactions of the individual intraocular muscles and (3) summarizing the effects of the more commonly used individual drugs on the pupil and on accommodation. Only those drugs having a peripheral action (that is, at the nerve ending or directly on the muscle itself) are discussed. Attention is principally restricted to those drugs which produce an effect when instilled into the conjunctival sac.

CHEMICAL MEDIATION OF NERVE IMPULSES

The concept of chemical mediation of nerve impulses has been developed in large measure by Loewi, by Dale and by Cannon and Rosenblueth. The following summary of this concept is taken in considerable part from the monograph by Cannon and Rosenblueth.[1]

The autonomic nervous system is divisible anatomically into the sympathetic and parasympathetic systems. It is also divisible, from the standpoint of chemical mediation at the nerve endings, into the adrenergic

From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

1. Cannon, W. B., and Rosenblueth, A.: Autonomic Neuro-Effector Systems, New York, The Macmillan Company, 1937.

and cholinergic systems. In general, the sympathetic system corresponds to the adrenergic and the parasympathetic to the cholinergic; exceptions are noted in the case of sweat glands, which are innervated by the sympathetic system but the nerve fibers of which have cholinergic endings, and in the case of certain blood vessels, the nerve supply of which is sympathetic but has a mixture of adrenergic and cholinergic endings.

CHOLINERGIC NERVOUS SYSTEM

The cholinergic nerve endings act through the liberation of acetylcholine, a substance which initiates the action of the innervated structure. Acetylcholine also transmits impulses in all ganglions (both sympathetic and parasympathetic), in the myoneural junctions of the voluntary nervous system and probably in all synapses of the central nervous system. Acetylcholine is rapidly destroyed in the body by a choline esterase, which is present in all tissues. It is destroyed so rapidly in the myoneural junctions of the voluntary nerves and in the synapses of the central nervous system that it can be detected only with difficulty after the passage of impulses at these points. The same applies to a lesser extent in the peripheral ganglions. However, at the autonomic endings of the cholinergic system its destruction is much slower, so that a certain amount diffuses into the surrounding tissue fluids and exerts an effect on neighboring cells. (Acetylcholine is normally present in the iris and ciliary body but is absent after degeneration of the parasympathetic system. It can also be detected in aqueous taken from physostigmine-treated rabbit's eye after exposure to light.[2]) This local diffusion is necessary in most parts of the cholinergic system in that there is only one nerve ending for a number of cells; notable exceptions are the intraocular muscles, which probably have as many nerve endings as they have muscle fibers.[3]

When acetylcholine is injected intravenously it is completely destroyed within a matter of seconds. Before it is destroyed it exerts a marked effect on all structures innervated by the parasympathetic system and a lesser effect on all autonomic ganglions but no demonstrable effect on the central nervous system or on voluntary muscles. A number of choline derivatives possess physiologic actions similar to those of acetylcholine. Most of these compounds, such as acetylbetamethylcholine (mecholyl), are inactivated almost (although not quite) as rapidly as is acetylcholine by the tissue esterase. The most important

2. Engelhart, E.: Humoraler Wirkungsmechanismus der Oculomotoriusreizung, Arch. f. d. ges. Physiol. **227**:220-234, 1931. Velhagen, K.: Zur Frage der vagotropen Substanzen im Auge, Arch. f. Augenh. **105**:573-602, 1932.

3. Clark, S. L.: Innervation of the Intrinsic Muscles of the Eye of the Cat, J. Comp. Neurol. **66**:307-325, 1937.

exception to this rule is carbaminoylcholine (doryl); the effects of this drug at the parasympathetic nerve endings are similar to those of acetylcholine, but it is inactivated only during the course of several hours.

The rapid destruction of choline compounds within the tissues may be inhibited by certain drugs with a carbamate (urethane) group (such as physostigmine or prostigmine) which combine with and inactivate the choline esterase. Acetylcholine is constantly being produced at all cholinergic endings as a result of normal parasympathetic "tonus"; thus the physiologic action of physostigmine or prostigmine is dependent on maintaining an abnormally high concentration of acetylcholine at the site of parasympathetic activity.

Pilocarpine and muscarine exert a direct effect on structures innervated by cholinergic nerves, having actions analogous to those of choline compounds in this respect. They are not destroyed by a choline esterase and consequently are not protected by physostigmine or prostigmine.

Atropine and its derivatives prevent the effects of choline compounds and of pilocarpine and muscarine on structures innervated by cholinergic nerves. However, atropine neither destroys these compounds nor renders the innervated structure unresponsive to stimulation with an electric current or to certain chemical compounds, such as histamine or barium chloride. Atropine does not prevent the effect of choline derivatives on the autonomic ganglions, nor does it inhibit the passage of impulses in the ganglions, in the voluntary myoneural junctions or in the central nervous system.

ADRENERGIC NERVOUS SYSTEM

The adrenergic nerve endings act through the liberation of epinephrine, which appears to be identical with the epinephrine liberated by the adrenal gland. Epinephrine, whether produced by the adrenal gland or by the nerve ending, will combine with either of two types of a hypothetic substance present in the vicinity of every cell innervated by the adrenergic system to form "sympathin." This substance (sympathin) in turn initiates the action of the innervated cell and of all adjacent cells to which it diffuses. Although there is only one type of epinephrine, there are two diametrically opposite types of sympathin, the type being dependent on which of the two types of hypothetic substance the epinephrine combines with. Sympathin E designates that type which produces excitation of structures innervated by adrenergic nerves, while sympathin I designates that type which causes inhibition of these structures. The action of any cell with an adrenergic innervation can be initiated either by stimulation of that particular nerve supply or by epinephrine reaching the cell through the general circulation (as after stimulation of the adrenal gland); also, the action which is initiated may in one instance be an excitation (as in the pupillary dilator and in the blood vessels of the

intestine, the liver and other organs) and in another instance an inhibition (as in the blood vessels of striated muscles and in the bronchiolar and intestinal walls).

Epinephrine and sympathin are both destroyed much more slowly than is acetylcholine. Diffusion of sympathin to neighboring cells plays a more prominent role in the adrenergic system, which has relatively few nerve endings per number of cells innervated, than does the diffusion of acetylcholine in the cholinergic system. If sufficient excitation of one branch of the adrenergic system takes place, remote effects are produced in other parts of the body by way of the blood stream. A stimulus such as pain or fright will cause reflex stimulation of the entire adrenergic system, whereas no physiologic stimulus will produce stimulation of the entire cholinergic system. Reactions of the adrenergic nervous system are usually much slower than those of the cholinergic system. For example, the pupil constricts more rapidly than it dilates.

It should be emphasized that impulses in all ganglions, adrenergic as well as cholinergic, are transmitted by means of acetylcholine. Thus the systemic administration of a large amount of acetylcholine, while primarily affecting structures with a cholinergic innervation, may also, by virtue of its effect on the adrenergic ganglions and by the liberation of epinephrine from the adrenal glands, induce some activity of adrenergic structures.

Experimentally, sympathin E may be liberated into the blood stream by stimulation of a branch of the sympathetic system innervating a structure normally excited by stimulation of the adrenergic nerves (for example, the liver).

Cells innervated by adrenergic nerves may be sensitized to the action of epinephrine compounds—i. e., may be caused to exhibit a greater quantitative reaction to a given amount of the epinephrine compound—by (1) the action of cocaine and certain other compounds, (2) a state of hyperthyroidism, (3) a state of pancreatic insufficiency and (4) recent postganglionic denervation (see under the section "Effect of Denervation").

A number of epinephrine derivatives possess qualitative actions somewhat similar to epinephrine but not exactly the same. Ephedrine probably acts directly on adrenergic structures to a limited extent, but at least the major portion of its effect is dependent on a cocaine-like action, sensitization of the structure to epinephrine. Other epinephrine derivatives intermediate between epinephrine and ephedrine with regard to the number of hydroxyl groups in the aromatic ring (such as neosynephrin, benzedrine and paredrine) are also intermediate in their pharmacologic mode of action.

There is no known substance which inhibits the action of sympathin or epinephrine in the sense that atropine compounds prevent the effects of acetylcholine. Ergotamine (ergotoxin or ergotamine tartrate), yohimbine and piperodimethylbenzodioxane (933F) in large doses inhibit the action of sympathin E on certain structures (especially on blood vessels), but they do not work in the case of certain other structures, and they also increase the tonus of smooth muscle in general.

EFFECT OF DENERVATION

Postganglionic section of any nerve results in an increased sensitivity of the innervated structure to the chemical mediator (sympathin or acetylcholine, as the case may be).[4] This increased sensitivity appears after a brief period of depression, reaches its maximum within several weeks, then gradually diminishes along with atrophy of the structure. The sensitivity will remain permanently elevated above the normal unless the nerve supply regenerates or unless the structure becomes very atrophic, and it is apparent in the reactions of the structure to certain epinephrine derivatives or to certain choline derivatives as well as to physiologic stimuli. Preganglionic section of any nerve results in an increased sensitivity of the ganglionic synapse and also of the innervated structure, but of less degree than that following postganglionic denervation.

REACTIONS OF THE INDIVIDUAL INTRAOCULAR MUSCLES

The iris and ciliary muscles are innervated by the autonomic nervous system. The principal sympathetic supply (postganglionic fibers arising from the cervical chain) reaches the eyeball through the long ciliary nerves, while the principal parasympathetic supply traverses the ciliary ganglion, where there is a synapse, and reaches the eyeball (postganglionic fibers) by way of the short ciliary nerves. It is possible that a small number of the sympathetic fibers pass through the ciliary ganglion and certain of the parasympathetic fibers do not pass through the ciliary ganglion, having their synapses in tiny subsidiary ganglions in the orbit.[3]

Stotler [5] and Clark [3] found that operative removal of the superior cervical sympathetic ganglion in the cat results in degeneration of fibers supplying the dilator muscle of the iris but not of those supplying the sphincter or ciliary muscles, while operative removal of the ciliary ganglion results in degeneration of only those fibers supplying the sphincter and ciliary muscles. However, these results are subject to some ques-

4. Cannon, W. B.: A Law of Denervation, Am. J. M. Sc. **198**:737-750, 1939.

5. Stotler, W. A.: Innervation of the Intrinsic Muscles of the Eye: An Experimental Study, Proc. Soc. Exper. Biol. & Med. **36**:576-577, 1937.

tion: Removal of all the ciliary ganglions or division of all the postganglionic sympathetic nerves cannot usually be accomplished, so that the histologic interpretation is somewhat complicated, and Clark mentioned that parasympathetic fibers in the ciliary muscle are so numerous that loss of a few other fibers (as after cutting the sensory supply) cannot be detected; this may explain why no loss of fibers is observed after cutting the cervical sympathetic nerves. Clark also noted that fibers from the cervical sympathetic nerves do enter the ciliary body, but he expressed the belief that they supply only the blood vessels. Physiologic evidence (see under the section "The Iris Muscles") indicates that the iris sphincter and the ciliary muscle may be partially innervated by cervical sympathetic nerves. While the principal nerve supply of the sphincter and ciliary muscles is undoubtedly parasympathetic, anatomic evidence does not appear to exclude the possibility of these muscles also having some sympathetic innervation.

THE IRIS MUSCLES

From a pharmacodynamic standpoint, the autonomic nerve supply of the iris muscles may be divided as follows: The dilator muscle is innervated solely by excitatory (sympathin E) adrenergic nerves. The sphincter muscle has a double innervation, being supplied with excitatory cholinergic nerves and also with inhibitory (sympathin I) adrenergic nerves. It is well known that stimulation of the ciliary ganglion causes contraction of the sphincter and that stimulation of the cervical sympathetic nerves produces contraction of the dilator. Joseph [6] removed the corneas of cats and dogs, separated part of the sphincter muscle from the dilator and found that stimulation of the cervical sympathetic nerves invariably caused prompt relaxation of the sphincter (which could be maintained by continued stimulation but disappeared when it was stopped). This experiment of Joseph, which unfortunately is reported in brief detail and has not been repeated by other investigators, affords the only direct physiologic evidence as to the existence of an inhibitory sympathetic nerve supply to the sphincter. However, strong indirect evidence of this innervation is furnished by the fact that the sphincter is relaxed by epinephrine and its derivatives (see subsequent text).

Both iris muscles have a normal tonus dependent on their nerve supply. Interruption of the sympathetic supply produces a relatively small pupil (part of Horner's syndrome), while interruption of the parasympathetic supply produces a semidilated pupil. Behr [7] has sup-

6. Joseph, D. R.: The Inhibitory Influence of the Cervical Sympathetic Nerve upon the Sphincter Muscle of the Iris, Am. J. Physiol. **55**:279-280, 1921.

7. Behr, C.: Der Anteil der beiden Antagonisten an der Pupillenbewegung bei den verschiedenen Reactionen, Arch. f. Ophth. **125**:147-155, 1930.

ported the theory that physiologic stimulation of the dilator is always accompanied by physiologic relaxation of the sphincter and vice versa. However, he has been unable to support this idea with any conclusive evidence. Observations by Gullberg and his co-workers,[8] using infrared photography to measure the size of rabbit pupils, are significant in this respect: After the oculomotor nerve is cut, the pupil becomes partially dilated and does not constrict to light or dilate in the dark, although it dilates to stimuli such as fear or pain and becomes smaller after also cutting the cervical sympathetic nerves. After division of the cervical sympathetic nerves alone, the pupil is smaller than normal but still constricts to light and partially dilates in dark adaptation. These observations indicate that the dilator muscle plays a passive role in the pupillary reaction to light, its tonus remaining constant, while the actual change in the size of the pupil is accomplished by a change in the tonus of the sphincter.

Much of the precise knowledge which has accumulated with regard to the iris muscles has arisen from in vitro experiments—that is, from preparing a freshly isolated segment of one of these muscles and observing the effect (by means of mechanical or photographic recording) of various solutions on its tonus. The first investigations of this sort were undertaken by Wessely.[9] His observations have been extended by numerous investigators, among whom may be mentioned Ten Cate,[10] Miller,[11] Poos,[12] Velhagen [13] and Heath and Geiter.[14] The transfer of data from these experiments to living eyes is subject to some criticism: The nerve supply of the muscles having been cut, there is a depression of the normal tonus. Also, these denervated muscles may exhibit an increased reaction to certain substances (such as epinephrine) and a decreased reaction to others (such as cocaine or physostigmine). How-

8. Gullberg, J. E.; Olmsted, J. M. D., and Wagman, J. I.: Reciprocal Action of the Constrictor and Dilator Pupillae During Light Adaptation, Proc. Soc. Exper. Biol. & Med. **38**:616-619, 1938.

9. Wessely, K.: Zur Frage der Existenz eines Dilatator iridis, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **42**:26-30, 1920.

10. Ten Cate, J., cited by Poos.[12a]

11. Miller, G. H.: The Effect of Cocaine on the Iris Compared with Its Effect on Certain Other Structures Containing Smooth Muscle, J. Pharmacol. & Exper. Therap. **28**:219-231, 1926.

12. Poos, F.: (*a*) Pharmakologische und physiologische Untersuchungen an den isolierten Irismuskeln, Arch. f. exper. Path. u. Pharmakol. **126**:307-351, 1927; (*b*) Die Funktionen der Irismuskeln im Lichte neurer Untersuchungen, Klin. Monatsbl. f. Augenh. **78**:227-238, 1927; (*c*) Ueber den Wirkungsmechanismus der Sympathikusreizmittel auf die isolierten Irismuskeln, ibid. **76**:5-14, 1926; (*d*) Zur Frage der Ergotaminwirkung am Auge, ibid. **79**:577-584, 1927.

13. Velhagen, K.: Ueber die Wirkungsweise von Cholinkorpern auf die Irismuskeln, zugleich ein Beitrag zur Frage der Doppelinnervation derselben, Arch. f. Augenh. **108**:126-136, 1934.

14. Heath, P., and Geiter, C. W.: Some Physiologic and Pharmacologic Reactions of Isolated Iris Muscles, Arch. Ophth. **21**:35-45 (Jan.) 1939.

ever, the qualitative reactions of isolated muscles should not differ from the reactions in the intact eye and are therefore of great importance. Most of the in vitro experiments have been performed on animal eyes, but the more essential results were verified by Poos with preparations from human eyes.

In vitro experiments have demonstrated that the dilator muscle contracts in the presence of epinephrine in high dilution (concentrations as low as 1 : 500,000,000—Poos). It also contracts in the presence of cocaine (concentrations as low as 1 : 100,000—Poos), ephedrine, neo-synephrin, benzedrine or paredrine. It is not affected by atropine, physostigmine, pilocarpine or morphine. Heath and Geiter noted that while the muscle is not affected by atropine alone, a relative relaxation is produced by atropine if the muscle is already contracted by epinephrine or neo-synephrin; this observation cannot readily be explained from a theoretic point of view. Poos observed no effect on the dilator muscle with choline, nor did Heath and Geiter with acetylcholine or with acetylbetamethylcholine. Velhagen, however, found that either acetylcholine or carbaminoylcholine produces slight contraction of the muscle if applied in sufficiently high concentrations (approximately the same concentration being necessary to produce this effect as is necessary to contract a fully atropinized sphincter muscle); he did not attach much significance to these reactions, because such high concentrations could hardly be attained in vivo. Ergotamine (in a concentration of 1 : 100,000 —Poos) produces a contraction, which may be increased by the addition of epinephrine.

The freshly isolated sphincter is contracted by choline, acetylcholine (concentrations as low as 1 : 400,000—Velhagen), carbaminoylcholine (concentrations as low as 1 : 400,000,000,000—Velhagen), physostigmine and pilocarpine. It is relaxed by atropine, scopolamine and homatropine. Relaxation of the sphincter is also produced by epinephrine (in dilutions as high as 1 : 500,000,000—Poos) and by cocaine (in dilutions as high as 1 : 1,000 according to Miller and as high as 1 : 10,000 according to Poos). Ephedrine, neo-synephrin and benzedrine also relax the sphincter. Heath and Geiter noted that a subreactive concentration of homatropine plus a subreactive concentration of epinephrine, neo-synephrin or benzedrine causes partial relaxation of a sphincter which is already contracted by acetylcholine (synergistic effect). Contraction of the sphincter is produced by ergotamine (1 : 100,000—Poos) and by morphine (1 : 15,000—Poos). Reaction to this high concentration of morphine is of little clinical significance, since this concentration could hardly be attained by systemic administration.

Several incidental in vitro actions of the iris muscles are worthy of mention. The tonus of both muscles is decreased by raising the temperature or by lowering the p_H. The tonus of both is increased by raising the oxygen tension of the solution and by potassium, barium or strontium

ions in $\frac{1}{100}$ molar solution. The tonus of the dilator is increased and that of the sphincter is decreased by calcium or magnesium ions (an effect similar to that of the epinephrine group of drugs). The dilator muscle of the rabbit responds to stimulation with the electrocurrent after an average latent period of 1.51 seconds, with a slow relaxation, whereas the latent period for the sphincter is only 0.52 second, with a rapid relaxation to less than the original tonus (Poos).

The relative parts played by relaxation of the sphincter and by contraction of the dilator after the instillation of epinephrine compounds is difficult to evaluate. The fact that maximum dilatation of the pupil with loss of the light reflex is often accomplished by strong solutions of an adrenergic drug suggests that there must be considerable relaxation of the stronger sphincter muscle. However, this relaxation is only relative, and the sphincter is not rendered insensitive to cholinergic excitation, because strong solutions of physostigmine will always either partially or completely counteract mydriasis produced by an adrenergic drug.

A few days after postganglionic paralysis of the cervical sympathetic nerves (Horner's syndrome) the pupil exhibits an increased sensitivity to epinephrine, which may persist indefinitely.[15] Dilatation of the pupil by cocaine in such an eye is greatly diminished; it is not known whether the slight dilatation caused by a strong solution is due to an effect on the denervated dilator muscle or on the sphincter. The actions of various epinephrine derivatives after sympathetic denervation are variable; Drake and his co-workers[16] observed that whereas there is a sensitization to epinephrine in both the cat and the rabbit, the mydriasis due to benzedrine (5 per cent) remains unchanged in the cat and that due to ephedrine (5 per cent) is prevented, while the opposite is true in the rabbit. Tsukamoto[17] found an increased sensitivity of the iris sphincter to pilocarpine two weeks after extirpation of the ciliary ganglion, and Shen and Cannon[18] noted a sensitization to acetylcholine, although Shaklee and Christensen[19] observed no increase in the reac-

15. Elliott, T. R.: (*a*) On the Action of Adrenalin, J. Physiol. **31**:20-21, 1904; (*b*) The Action of Adrenalin, ibid. **32**:401-467, 1905.

16. Drake, M. E.; Renshaw, R. J. F.; Modern, F. S., and Thienes, C. H.: The Smooth Muscle Actions of Epinephrine Substitutes: VII. Responses of Denervated Smooth Muscles of Iris and Intestine to Epinephrine, Ephedrine, Amphetamine (Benzedrine) and Cocaine, J. Pharmacol. & Exper. Therap. **66**:251, 1939.

17. Tsukamoto, cited by Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1, p. 545.

18. Shen, S. C., and Cannon, W. B.: Sensitization of Denervated Pupillary Sphincter to Acetylcholine, Chinese J. Physiol. **10**:359-371, 1936.

19. Shaklee, A. O., and Christensen, K.: Action of Carbaminoylcholine on Iris, Normal and Parasympathectomized, Proc. Soc. Exper. Biol. & Med. **32**:1619-1620, 1935.

tion to carbaminoylcholine. Physostigmine has no effect on the pupil after complete degeneration of the parasympathetic nerves,[20] since no acetylcholine is being formed in such an eye.

THE CILIARY MUSCLE

The mode of action of the ciliary muscle is not completely clear. Conclusive evidence has not as yet been offered as to whether it has a single or dual nerve supply, nor whether (if it has a dual supply) it is a single muscle or two closely intertwined antagonistic muscles. However, contraction of at least the major portion of the ciliary muscle is induced by parasympathetic (cholinergic) nerves, and such contraction results in relaxation of the zonule with an increase in the equatorial diameter of the lens.

The mechanism of accommodation has been extensively studied since the time of Helmholtz; a complete summary of the collected evidence exists in the recent monograph by Fincham.[21] The studies of Fincham indicate that the original theory set forth by Helmholtz is essentially correct: Relaxation of the ciliary muscle causes increased tension of the zonule, with flattening of the lens and consequent accommodation for distance. Contraction of the ciliary muscle relaxes the zonule and permits the lens to assume a more globular shape, with a hyperboloid anterior surface and consequent accommodation for near vision. The inherent tendency of the lens to assume a globular shape apparently depends on the shape, thickness and elasticity of the lens capsule rather than on the cortical substance (as originally postulated by Helmholtz).

Any drug which enhances or simulates the action of acetylcholine (physostigmine, pilocarpine and others) will produce contraction of the ciliary muscle, and atropine or any of its derivatives will cause relaxation. The degree of contraction or relaxation depends on the individual drug and its concentration.

The probability of dual innervation of the ciliary muscle, the sympathetic system effecting accommodation for distance and the parasympathetic system accommodation for near vision, was first suggested by Morat and Doyon in 1891. Considerable evidence has accumulated in support of this concept, mainly as a result of the investigations of Poos [22] and of Cogan.[23] The best part of this evidence may be summarized as follows:

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20. Anderson, H. K.: The Action of Eserine and Atropine upon the Denervated Sphincter Iridis, J. Physiol. **31**:22-24, 1904.

21. Fincham, E. F.: The Mechanism of Accommodation, Brit. J. Ophth., 1937, supp. 8, pp. 5-80.

22. Poos, F.: Zur Frage der sympathischen Innervation des Ziliarmuskels und ihrer Bedeutung für die Akkommodation, Klin. Monatsbl. f. Augenh. **80**:749-766, 1928.

23. Cogan, P. G.: Accommodation and the Autonomic Nervous System, Arch. Ophth. **18**:739-767 (Nov.) 1937.

1. Poos found that glaucosan (a strong synthetic epinephrine preparation) dropped into the conjunctival sac of normal eyes produced considerable paresis of accommodation, lasting around forty-five minutes. The average reduction of accommodation was about 3.5 diopters. Strong solutions of cocaine (10 per cent) and ephedrine (10 per cent) each produced similar results. The near points of accommodation were determined by utilization of Scheiner's experiment, which is a procedure subject to relatively small errors. Cogan observed that the subconjunctival injection of a 0.1 per cent solution of epinephrine reduced the amplitude of accommodation 1.5 and 2.5 diopters (two experiments) within twenty minutes. He controlled the size of the pupil with a 2 mm. diaphragm and measured the near point of accommodation with test type. Gradle [24] observed a reduction of accommodation to approximately one half within twenty-five to forty-five minutes after the application of a 0.1 per cent solution of epinephrine beneath the upper lid by means of a cotton pledget; details of his observations are not given.

2. Cogan examined 5 patients with Horner's syndrome of recent onset and in each case found a greater amplitude of accommodation on the affected side than on the normal side. The average difference was about 1.5 diopters.

3. Poos found in a patient with Horner's syndrome that paresis of accommodation could be brought about on the affected side by a small amount of epinephrine but not by a strong concentration of cocaine. This phenomenon is to be expected after postganglionic paralysis of an adrenergic innervated structure.

4. Myerson and Thau [25] stated that benzedrine produced paresis of accommodation but give no details of their observations. Equivocal results obtained by other authors using benzedrine in low concentration (1 per cent) are mentioned later. Heath [26] found that repeated instillations of neo-synephrin caused partial loss of accommodation, but he did not state the quantitative reduction or the method of determination.

Poos attempted to study the in vitro actions of the ciliary muscle, but he was mechanically unable to measure the effects of either stimulation with the electric current or of drugs on the muscle.

Some evidence exists which is unfavorable to the concept of dual innervation of the ciliary muscle. The anatomic investigations of Stotler

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24. Gradle, H. S.: The Use of Epinephrine in Ocular Hypertension, J. A. M. A. **84**:675 (Feb. 28) 1925.

25. Myerson, A., and Thau, W.: Human Autonomic Pharmacology: IX. Effect of Cholinergic and Adrenergic Drugs on the Eye, Arch. Ophth. **18**:78-90 (July) 1937.

26. Heath, P.: Neosynephrin Hydrochloride: Some Uses and Effects in Ophthalmology, Arch. Ophth. **16**:839-846 (Nov.) 1936.

and of Clark have already been mentioned, and Strebel [27] was unable to demonstrate a biphasic action current in the muscle. Also, Lokshin and Sherman [28] observed no change in accommodation in 20 patients after production of a temporary Horner's syndrome by block of the cervical sympathetic nerves. However, it appears certain that adrenergic drugs affect the ciliary muscle to an appreciable degree, whether or not the muscle possesses a physiologic adrenergic innervation.

Two radically different ideas exist as to how the adrenergic innervation (if it exists) opposes the principal cholinergic action of the ciliary muscle. Contraction of the circular fibers obviously relaxes the zonule, and the longitudinal (meridional) fibers apparently produce the same effect by shortening the base of the triangular muscle. Contraction of radial fibers, however, can conceivably act in the opposite way, tending to increase zonular tension. The muscle fibers are so intertwined that it is doubtful whether a group of purely "radial" fibers exists, but Cogan suggested that they may exist as a physiologically separate muscle, innervated by sympathetic nerves and acting as an antagonist to the circular and longitudinal fibers. Poos, on the other hand, supported the hypothesis that the entire muscle is constricted by cholinergic stimulation but is relaxed by adrenergic stimulation. As an analogy, he pointed out that the muscular walls of the intestine and bronchioles, the iris spincter and certain other structures are constricted by cholinergic nerves and relaxed by adrenergic nerves. All of the evidence in favor of dual innervation of the muscle is explicable by either of these hypotheses, but Clark's [3] observation that every part of the ciliary muscle receives approximately the same number of parasympathetic fibers appears to lend support to the idea of Poos.

EFFECTS OF VARIOUS DRUGS ON THE PUPIL AND ON ACCOMMODATION

Drugs introduced into the general circulation reach the intraocular muscles only in a concentration equal to that present in other parts of the body. Every compound which affects these muscles also has a profound effect on other structures and is potentially toxic if administered in sufficiently large doses. Only relatively slight reaction of the intraocular muscles is obtained after systemic administration of subtoxic doses of these drugs.

Solutions dropped into the conjunctival sac diffuse through the cornea and from the aqueous into the iris and ciliary muscles. Diffusion into

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27. Strebel, J.: Ueber die Mechanik des Akkommodationsvorganges und die Wirkungsweise der drei Fasersysteme des Ziliarmuskels zur Widerlegung der Diskontinuitatstheorien, Klin. Monatsbl. f. Augenh. **95**:235-246, 1935.

28. Lokshin, S. Y., and Sherman, L. M.: Horner's Syndrome and Disturbance of Ocular Accommodation, Vestnik oftal. (no. 1) **15**:60-63, 1939; abstracted, Am. J. Ophth. **23**:226, 1940.

the general circulation takes place only to a limited extent, mostly by way of the tear passages and nasopharynx. A profound effect may thus be obtained on the intraocular structures with little effect on other parts of the body.

It must not be assumed that general toxic effects never occur, because they do. Enough general absorption occurs with repeated instillations of any potent solution that some remote effects are observed, and in addition a few persons are highly susceptible to the effects of certain drugs. In instances in which a maximum effect on the ocular structures is desired the possibility of toxic reactions should always be borne in mind. Toxic reactions are most often encountered (in the approximate order of occurrence) with scopolamine, cocaine, atropine, physostigmine and epinephrine. Another factor which determines the usefulness of a drug in instances in which it must be used over a long period is the occurrence of local irritation or of hypersensitivity. Undue local irritation is most frequently encountered with physostigmine; hypersensitivity may appear after the use of any of these drugs but is most frequent in the case of the atropine series.

The cornea is the principal barrier through which a drug must pass in order to reach the muscles, and the length of time a solution remains in contact with the cornea determines in large part the amount which diffuses through. Heath [29] advised the use of water-in-oil emulsions instead of aqueous solutions in order to obtain greater and more even absorption. According to Mestrezat and Morax,[30] anions pass through the cornea more easily than cations, and both pass through more easily in weakly acid solution. The condition of the corneal epithelium greatly influences the permeability of the cornea. Thus, solutions diffuse into the aqueous much more readily after an abrasion of the cornea (easily demonstrated in the case of fluorescein) or after the use of cocaine.

Drugs reach the ciliary muscle more slowly and in smaller concentration than they do the iris muscles, because the circulation of aqueous is normally away from the ciliary body. For example, the minimal amount of atropine instilled into the conjunctival sac which will depress accommodation is said to be ten to fifteen times that amount which will produce an effect on the pupil.[31]

Most mammalian eyes exhibit the same qualitative reactions to "miotics" and "mydriatics," but there are certain confusing exceptions

29. Heath, P.: Use of Emulsions in Ophthalmology, Am. J. Ophth. **22**:904-905, 1939.

30. Mestrezat and Morax, cited by Grafe.[31]

31. Grafe, E.: Pharmakologische Wirkungen auf Iris und Ciliarmuskel, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1929, vol. 12, pt. 1, pp. 196-232.

(as in the case of pilocarpine and of ergotamine), and the quantitative reactions vary considerably in different species. In human beings these drugs usually affect the muscles of lightly pigmented eyes more readily than those of heavily pigmented eyes, [32] and individual quantitative variation is great.

In general, then, a drug dropped into the conjunctival sac will affect the intraocular muscles with little effect on other bodily structures; it will reach the iris muscles sooner and in higher concentrations than it will the ciliary muscle, and the concentrations which reach the muscle will vary greatly with the species, the individual and the type of solution.

Subconjunctival injection of a drug leads to diffusion of the drug through the anterior part of the sclera and periphery of the cornea. This method of administration generally results in more rapid absorption and a higher intraocular concentration than does instillation into the conjunctival sac, but at the same time more of the drug is absorbed into the general circulation, with more likelihood of a toxic reaction.

CHOLINE COMPOUNDS

Acetylcholine.—This drug instilled into the conjunctival sac in concentrations of 5 to 20 per cent usually produces a rather slight transient miosis (lasting only a few minutes) in rabbits or cats. Individual variation is marked, and sometimes an appreciable effect may be obtained with concentrations as low as 1 per cent. Molliter [33] observed that in rabbits the miotic effect is considerably enhanced by massage of the lids after instillation. (He also made this observation in the case of several other choline compounds and of physostigmine.) The only available report of the miotic effect of this drug in human beings is that of Passow,[34] who noted that a 1 per cent solution usually has no effect but occasionally produces a slight, transient pupillary constriction. Injected subconjunctivally, acetylcholine is more certain in its action, eliciting a minimal miosis (in rabbits) with a dose as low as 0.01 Gm. and producing maximal miosis within one minute with a dose of 0.12 Gm.[35] No reports of the effect of this drug on accommodation are available.

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32. Chen, K. K., and Poth, E. J.: Racial Differences as Illustrated by the Mydriatic Action of Cocaine, Euphthalmine and Ephedrine, J. Pharmacol. & Exper. Therap. **36**:429-445, 1929.

33. Molliter, H.: A Comparative Study of the Effects of Five Choline Compounds Used in Therapeutics, J. Pharmacol. & Exper. Therap. **58**:337-360, 1936.

34. Passow, A.: Ueber Beziehungen des Gesamtorganismus zum primären Glaukom, Arch. f. Augenh. **103**:111-160, 1930.

35. (*a*) Villaret, M.; Justin-Besançon, L.; Schiff-Wertheimer (Mme.), and Gallois, J.: Les esters de la choline en ophtalmologie, Arch. d'opht. **49**:129-165, 1932. (*b*) Courland, M., and Kahane, E.: Action de l'acétylcholine sur l'œil du lapin, Compt. rend. Soc. de biol. **96**:1136, 1928.

Acetylbetamethylcholine.—Instillation of this drug into the conjunctival sac of human beings in concentrations of 1 to 20 per cent generally causes marked miosis, depending on the individual susceptibility.[25] A similar effect is noted in cats and rabbits.[36] The minimal concentration necessary to produce miosis in the rabbit was found by Molliter to be 0.1 per cent. The miosis reaches its maximum in four to twenty minutes and may persist as long as two hours. Myerson and Thau [25] stated that in human beings "invariably with sufficient doses the near point is diminished, and the ability to read at that point improved."

Carbaminoylcholine.—Instillation of this drug into the conjunctival sac of human beings in concentrations of 0.5 to 2 per cent produces marked miosis, beginning in ten to twenty minutes and persisting from three to eight hours.[37] The miotic effect is variously stated to be greater than, equal to or less than that of pilocarpine—the degree of miosis is probably greater with carbaminoylcholine than with pilocarpine of equal strength, but the effect is not as prolonged. Molliter observed minimal constriction of the pupil in rabbits with concentrations as low as 0.01 per cent. No observation as to its effect on accommodation can be found.

PILOCARPINE

Pilocarpine Hydrochloride.—This drug instilled into the conjunctival sac in a 1 to 3 per cent solution produces a marked (but usually not maximal) miosis, beginning after about ten minutes and completely disappearing within twenty-four hours.[38] The concentration necessary to elicit a minimal pupillary constriction in human beings is said to be 0.02 to 0.25 per cent.[39] When the effect of the drug wears off a slight transient mydriasis appears, indicative of partial paresis (or decreased

36. Hunt, R.: Notes on Acetyl-Methyl-Choline, J. Pharmacol. & Exper. Therap. **55**:268-273, 1935. Molliter.[33] Villaret and others.[35a]

37. Velhagen, K., Jr.: Die praktische Verwendbarkeit des Doryls (Carbaminoylcholin) in der Glaukombehandlung, Klin. Monatsbl. f. Augenh. **92**:472-483, 1934. Miloro, A.: L'azione del doryl nella cura del glaucoma, Ann. di ottal. e clin. ocul. **63**:780-793, 1935; abstracted, Zentralbl. f. d. ges. Ophth. **36**:29, 1936. Wilenkin, M.: Ueber die Wirkung des Doryls auf den intraokulären Druck und Pupillenweite, Klin. Monatsbl. f. Augenh. **96**:84-90, 1936. de Sanctis, G. E.: L'azione del doryl sull'occhio in condizioni normali e in case di glaucoma, Ann. di ottal. e clin. ocul. **65**:25-37, 1937. Magitot, M. A.: Les corps choliniques comme collyres hypotenseurs, Bull. Soc. d'opht. de Paris **5**:38-41, 1939.

38. Bing, R., and Franceschetti, A.: Die Pupille (Pharmakologischer Teil), in Schieck, F., and Bruckner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 6, pp. 113-120.

39. Marui, S.: Ueber Kombinationswirkung von Physostigmin (Eserin) und Pilokarpin am menschlichen Auge, Klin. Monatsbl. f. Augenh. **68**:145-152, 1922.

reactivity) of the sphincter.[40] There are other indications that pilocarpine decreases the contractile activity of the sphincter: In the rat the effect is entirely paretic, pilocarpine invariably dilating the pupil (acting as an antagonist to physostigmine and a synergist to cocaine or epinephrine).[41] In the rabbit pilocarpine produces relatively slight miosis, which cannot be increased by repeated instillations; after several instillations the light reflex is abolished and the reactivity to physostigmine is depressed (personal observations). Since the rabbit dilator is not affected by pilocarpine, this effect can be ascribed only to reduced contractibility of the sphincter. Pilocarpine produces a partial, but usually not marked, contraction of the ciliary muscle; this effect is said also to be followed by partial paresis of the ciliary muscle.[31]

PHYSOSTIGMINE AND PROSTIGMINE

Physostigmine Salicylate.—Instillation of this drug into the conjunctival sac of human beings in concentrations of 0.1 to 1 per cent produces maximal miosis and a spasm of accommodation, beginning within ten minutes and reaching its maximum after about thirty minutes. The intense accommodative spasm wears off after about two hours, but for some time afterward accommodation for near vision remains hyperexcitable, so that the slightest effort to focus for near vision results in ciliary spasm. Intense miosis persists around twelve hours, and some degree of miosis (with increased irritability of the sphincter to stimuli such as light or accommodation) is present for one or more days. The concentration of physostigmine necessary to elicit minimal pupillary constriction in human beings is said to be about 0.01 per cent.[39] In rabbits, 1 drop of a 0.001 per cent solution consistently produces miosis.[33]

Prostigmine Bromide.—Instillation of prostigmine bromide into the conjunctival sac of human beings in a 3 to 5 per cent solution produces maximal miosis and spasm of accommodation, beginning within a few minutes and persisting for a number of hours. The qualitative actions of this drug on the eye appear to be exactly similar to those of physostigmine, but a quantitative comparison is difficult to evaluate from the available data. Myerson and Thau[25] stated that the minimal concentration of prostigmine effective in constricting the pupil is a 1 per cent solution, but Rossi[42] obtained an appreciable miosis in human beings

40. Harnack, E., and Meyer, H.: Untersuchungen über die Wirkungen der Jaborandi-Alkaloide nebst Bemerkungen über die Gruppe des Nicotins, Arch. f. exper. Path. u. Pharmakol. **12**:366-400, 1880.

41. Waddell, J. A.: The Action of Pilocarpin on the Rat's Pupil, J. Lab. & Clin. Med. **12**:232-239, 1926.

42. Rossi, G.: Azione sull'occhio di un nuovo farmaco sintetico eserinosimile: la prostigmina, Arch. di ottal. **42**:341-360, 1935.

with several instillations of a 0.05 per cent solution. Rossi also noted that in rabbits a 0.5 per cent solution of prostigmine is approximately equivalent to the same strength of physostigmine, with regard to the miotic effect. Clarke [43] is of the opinion that prostigmine is approximately equal to physostigmine from the standpoint of effect produced but that it can be used in about twice as strong concentration as physostigmine can without causing irritation.

ATROPINE COMPOUNDS

Atropine Sulfate.—Instillation of atropine sulfate into the conjunctival sac of human beings in a 1 per cent solution produces marked mydriasis (but not maximal, since the iris dilator is not stimulated), beginning within fifteen minutes and gradually subsiding during a period of about ten days. Cycloplegia of somewhat variable degree, but usually relatively complete, begins to appear within twenty-five minutes and passes off after three to five days. [31] Cycloplegia is more consistently complete after multiple instillations but also persists for a longer period. The minimal concentration of atropine which will produce recognizable mydriasis is said to vary from 1 in 100,000 to 1 in 50,000,000, less being required in adults than in infants.[44]

Homatropine Hydrobromide.—This drug has a qualitative effect on the intraocular muscles similar to that of atropine, but the quantitative effect is weaker and of shorter duration. In concentrations of 1 or 2 per cent a single instillation produces mydriasis, beginning within fifteen minutes and persisting approximately twenty-four hours; cycloplegia is usually not complete and passes off before the mydriasis does. Repeated instillations of a 1 or 2 per cent solution produce relatively complete cycloplegia, which completely disappears within twenty-four to forty-eight hours; a single instillation of a 5 per cent solution of homatropine induces cycloplegia of almost the same degree but of shorter duration, lasting an average of about twenty-four hours.[45]

Scopolamine Hydrobromide.—This drug in the form of its levorotatory isomer (hyoscine) similarly dilates the pupil and paralyzes accommodation. Quantitatively, it is about ten times as powerful as atropine, whereas its effect is of shorter duration. One drop of a 0.1 to 1 per

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43. Clarke, S. T.: Mecholyl and Prostigmin for Glaucoma, Am. J. Ophth. **22**:249-257, 1938.

44. Naito, I.: Ueber Kombinationswirkung von Atropin und Kokain am menschlichen Auge, Klin. Monatsbl. f. Augenh. **68**:153-160, 1922. Bing and Franceschetti.[38]

45. (*a*) Sudranski, H. F.: An Evaluation of Homatropine-Benzedrine Cycloplegia, Arch. Ophth. **20**:585-596 (Oct.) 1938. (*b*) Thorne, F. H., and Murphey, H. S.: Cycloplegics, ibid. **22**:274-287 (Aug.) 1939.

cent solution produces mydriasis within ten minutes, lasting two to seven days, and relatively complete cycloplegia, passing off after about two days. The racemic (optically inactive) form of scopolamine (atroscine) is only about one-half as active as the levorotatory form.

The relative degree of cycloplegia produced by atropine, scopolamine and homatropine is a question of considerable practical importance. Bothman [46] examined a large number of eyes of young patients after instillation of each of these drugs and by noting the differences in refraction determined the following quantitative relation: On the average, the residual accommodation with scopolamine (two instillations of a 0.5 per cent solution with thirty minute intervals) was 0.16 diopter greater than that with atropine (fourteen instillations of a 1 per cent solution, administered four times a day); the residual accommodation with homatropine (six instillations of a 2 per cent solution with ten minute intervals) was 0.49 diopter greater than that with atropine or 0.33 diopter greater than that with scopolamine. These values can only be taken as a broad average, because there was considerable individual variation.

Atropine Methylnitrate (Eumydrine) and Atropine Methylbromide (Mydriasine).—These drugs in a 1 to 5 per cent solution have qualitative effects similar to those of atropine; quantitatively, their action lies almost midway between that of atropine and that of homatropine. Mydriasis produced by these drugs wears off within about five days.

Duboisine Sulfate and Hyoscyamine.—These two drugs, chemically the same, although obtained from different sources, in a 0.5 to 1 per cent solution have a qualitative and quantitative effect on the intraocular muscles similar to that of scopolamine.

Euphthalmine Hydrochloride.—This drug has the weakest and most evanescent action of any widely used drug in the atropine group. One instillation of a 5 or 10 per cent solution produces mydriasis lasting five to ten hours but has only a minimal effect on accommodation.

EPINEPHRINE COMPOUNDS

Epinephrine Hydrochloride or Bitartrate.— These drugs instilled into the conjunctival sac of human beings in a 0.1 per cent solution are very inconstant in their action, usually having no effect on the pupil but sometimes producing a transient mydriasis. Knapp [47] observed that repeated instillation of a 0.1 per cent solution of epinephrine produces

46. Bothman, L.: Refractive Errors in the Same Eyes While Under the Influence of Homatropin, Scopolamin, and Atropin, Tr. Am. Acad. Ophth. **42**: 165-178, 1937.

47. Knapp, A.: The Action of Adrenalin on the Glaucomatous Eye, Arch. Ophth. **50**:556-559, 1921.

mydriasis in a high percentage of eyes of glaucomatous patients. If a 0.1 per cent concentration is applied by means of a cotton wick inserted beneath the upper lid [24] or if this concentration is injected subconjunctivally, maximal mydriasis usually appears within several minutes and persists for several hours. In the form of a 1 per cent ointment [48] or a 1 to 2 per cent solution,[49] conjunctival instillation of epinephrine results in mydriasis of marked degree; according to Horner and Bettman, maximal mydriasis (with 1 drop of a 2 per cent solution) is maintained in elderly patients for about two hours, while some degree of mydriasis persists for an average of eight hours. (The mydriatic effect of epinephrine is generally less marked in the young than in the aged.) Epinephrine sometimes abolishes the light reflex but more often only diminishes it. The experiments of Poos (using a 2 per cent solution by instillation), of Cogan (using a 0.1 per cent solution subconjunctivally) and of Gradle (using a 0.1 per cent solution in a cotton pledget beneath the upper lid), in which a reduction of accommodation was demonstrated, have already been cited.

Synthetic Epinephrine.—Epinephrine in the form of the levorotatory isomer is most commonly prepared commercially as a chloride or bitartrate salt, either by extraction from the adrenal glands of animals or by synthesis. Epinephrine is available in the form of a 1 per cent ointment, as a powder suitable for the preparation of strong solutions or in 0.1, 1 and 2 per cent solutions. Epinephrine oxidizes during the course of several weeks when exposed to air. It can be preserved for long periods sealed in air-tight containers without oxygen, and it is relatively stable in a solution of low p_H which also contains a reducing agent. Solutions of an epinephrine salt without a reducing agent must be prepared shortly before use; the commercial solution (and ointments such as 1 per cent suprarenin bitartrate in a water-soluble base) are relatively stable, deteriorating only during the course of months. The laevo-glaucosan of Hamburger is a solution of 2 per cent laevo-epinephrine plus 2 per cent methylaminoacetopyrocatechol (a precursor in the synthesis of epinephrine, possessing almost no adrenergic action). This compound is expensive and has an action practically identical with that

48. Thiel, R.: Zur Wirkung des Adrenalins auf dem Binnendruck des Glaukomauges, Klin. Monatsbl. f. Augenh. **72**:534, 1924.

49. (*a*) Hamburger, C.: Treatment of Glaucoma with Glaucosan, Glaucosan Drops, and Amine-Glaucosan Drops, Arch. Ophth. **55**:533-544, 1926. (*b*) Green, J.: Two Per Cent Epinephrine Solutions as Substitutes for Levo-Glaucosan, ibid. **5**:350-353 (March) 1931. (*c*) Horner, W. D., and Bettman, J.: Synthetic Suprarenin Bitartrate as a Mydriatic, Am. J. Ophth. **19**:311-315, 1936. (*d*) Post, L. T.: Comparison of Suprarenin Bitartrate and Cocaine Plus Euphthalmine as Mydriatics for Elderly Patients, ibid. **20**:33-39, 1937. (*e*) Howell, S. C.: Action of Epinephrine on Diseased Human Eye, Arch. Ophth. **16**:1018-1027 (Dec.) 1936.

of any 2 per cent solution of epinephrine. The dextro-glaucosan of Hamburger consists of 1 part dextro-epinephrine (which is only about one-fifteenth as active as laevo-epinephrine) plus five parts methylamino-acetopyocatechol. This compound is not often used today; it was formerly administered by subconjunctival injection in solution such that there was 0.2 per cent dextro-epinephrine or by conjunctival instillation in much more concentrated solution. The amine-glaucosan of Hamburger must not be confused with the foregoing drugs; this is a strong solution of histamine (see under section, "Histamine").

Neo-Synephrin Hydrochloride.—This drug instilled into the human eye in a 0.25 to 2 per cent solution produces marked (often maximal) dilatation of the pupil, beginning within ten minutes and lasting from one and a half to eight hours.[50] The light reflex may be abolished but is usually partially retained. Solutions as strong as 10 per cent (in the form of an emulsion) have been used by Heath for breaking resistant posterior synechiae. Heath noted little effect on accommodation with a single instillation of a 1 per cent solution but stated that repeated instillations (4 to 6 drops) caused partial loss of accommodation for from two to ten hours (method of measuring accommodation not given). Neo-synephrin appears to be approximately equal to epinephrine in point of view of quantitative effects on the intraocular muscles, except that the effects are somewhat more prolonged.

Benzedrine Sulfate.—Instillation of this drug into the conjunctival sac in a 0.25 to 1 per cent solution produces mydriasis in from fifteen to thirty minutes, lasting several hours. The light reflex is diminished but does not disappear. Solutions as strong as 10 per cent have been used. The statement of Myerson and Thau that benzedrine reduces the ability to accommodate has already been mentioned. (No details as to the strength of solution or as to the measurement of accommodation are given.) Sudranski [45a] was unable to demonstrate any diminution of accommodation in 15 eyes after the instillation of 1 drop of a 1 per cent solution of benzedrine; he determined the near point of accommodation and controlled the size of the pupil with a 3 mm. pinhole. Thorne and Murphey [45b] stated that three instillations of a 1 per cent solution of benzedrine induced an average maximum depression of accommodation of 0.75 diopter within sixty minutes, but they did not mention compensating for the increased pupillary diameter by means of uniform pinhole. Weinman and Fralick [51] observed a decrease in accommodation of about 3 diopters after instilling a 1 per cent solution of benzedrine, but again there is no mention of compensating for the pupillary diameter.

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50. Post, W. L.: Neo-Synephrine, Am. J. Ophth. **20**:170-177, 1937. Heath.[26]

51. Weinman, E. B., and Fralick, F. B.: Comparative Study of Benzedrine, Paredrine, and Cocaine with Homatropine as Cycloplegics, Am. J. Ophth. **23**:172-178, 1940.

Paredrine Hydrobromide [52].—This drug instilled into the conjunctival sac in a 0.15 to 2 per cent solution produces mydriasis beginning within ten to thirty minutes, reaching a maximum within about sixty minutes and lasting around six hours.[53] Abbott and Henry stated that no loss of accommodation was observed with paredrine in a series of 22 patients; the method of measuring accommodation is not given. Thorne and Murphey observed the same effect on accommodation with paredrine as with benzedrine (an average maximum depression of 0.75 diopter after three instillations of a 1 per cent solution). This observation is subject to the same criticism as that mentioned in the case of benzedrine, namely, that no mention is made of compensating for the increased pupillary diameter.

Ephedrine Sulfate.—Instillation of this drug into the human eye in a 10 per cent solution produces a slight to moderate mydriasis, beginning within fifteen to thirty minutes and persisting three to nine hours. The light reflex remains active. The decreased accommodation noted by Poos following the use of ephedrine has already been mentioned. Chen and Path [54] also noticed a decrease in accommodation (of slightly more than 1 diopter), but they apparently did not compensate for the mydriasis while determining the near point.

COCAINE

Cocaine.—Instillation of cocaine into the conjunctival sac in a 2 to 10 per cent solution produces slight to maximal mydriasis beginning within ten minutes, reaching its maximum within about forty-five minutes and disappearing after an average of about four hours. Horovitz [55] found an average reduction in accommodation of 3 diopters following 1 drop of a 6 per cent solution and practically complete paralysis of accommodation after six instillations of a 6 per cent solution. The reduction in accommodation observed by Poos has already been mentioned. The maximal reduction of accommodation is attained approximately thirty minutes after instillation of the drug, lasts about fifteen minutes and rapidly disappears during the following hour.

The mechanism of the motor action of cocaine is not known, beyond the fact that it sensitizes structures to the action of epinephrine. The

52. This is a proprietary name for a sympathomimetic drug (4-hydroxy-α-phenyl-β-aminopropane; β-4-hydroxyphenyl isopropylamine.

53. (*a*) Abbott, W. O., and Henry, C. M.: Paredrine (β-4-Hydroxyphenylisopropylamine); Clinical Investigation of Sympathomimetic Drug, Am. J. M. Sc. **193**:661-673, 1937. (*b*) Tassman, I. S.: The Use of Paredrine in Cycloplegia, Am. J. Ophth. **21**:1019-1024, 1938.

54. Chen, K. K., and Path, E. J.: Ephedrine as a Mydriatic in Caucasians, Am. J. M. Sc. **178**:203-209, 1929.

55. Horovitz, I.: Der Einfluss von Kokain und Homatropin auf Akkommodation und Pupillengrosse, Ztschr. f. Augenh. **28**:530-538, 1912.

paralyzing effect it has on sensory terminals is apparently not related to this action, because most local anesthetics do not have a motor action. A few cocaine derivatives, such as tutocain and tropacocaine, produce a slight to moderate mydriasis.

HISTAMINE

Histamine.—Instillation of this drug into the conjunctival sac in a 2 to 10 per cent solution or injection subconjunctivally in a dose of 0.0002 Gm. produces an intense miosis, beginning within several minutes and persisting for several hours. Its effect is not opposed by the action of atropine. Hamburger [49a] stated that histamine in strong solution is the most powerful miotic known. Unfortunately, it also causes considerable chemosis and inflammation of the conjunctivas and lids and is painful unless preceded by a local anesthetic. Also, it lowers the blood pressure appreciably when used in a 10 per cent solution. A report of the effect of histamine on accommodation is not available.

ERGOTAMINE

Ergotamine Tartrate.—This drug, essentially identical with ergotoxin,[56] is of interest because its use has been proposed by various authors during recent years (on purely theoretic grounds) as a miotic which will act by paralyzing the adrenergic action of the iris dilator. This idea is the result of the experiments of Dale [57] and others demonstrating that ergotamine administered systemically inhibits the excitatory action of epinephrine on blood vessels and constricts the pupil of the cat. However, ergotamine dilates the pupil of the rabbit; and the in vitro observations of Poos that ergotamine increases the tonus of both sphincter and dilator (without inhibiting the action of epinephrine) have already been cited. Thiel [58] and Heim [59] have utilized ergotamine subcutaneously and orally in the treatment of glaucoma; while they considered the effects on glaucoma favorable, they noted either no change in the size of the pupil or a minimal miosis (hardly 0.5 mm. constriction—Thiel).

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56. Dale, H. H., and Spiro, K.: Die wirksamen Alkaloide des Mutterkorns, Arch. f. exper. Path. u. Pharmakol. **95**:337-350, 1922.

57. Dale, H. H.: (*a*) Action of Ergot, J. Physiol. **34**:163-206, 1906; (*b*) On the Action of Ergotoxin with Special Reference to the Existence of Sympathetic Vasodilators, ibid. **46**:291-300, 1913.

58. Thiel, R.: Experimentelle und klinische Untersuchungen über den Einfluss des Ergotamins (Gynergens) auf den Augendruck beim Glaukom, Klin. Monatsbl. f. Augenh. **77**:753, 1926.

59. Heim, H.: Zur Behandlung des Glaukoms mit Ergotamin, Klin. Monatsbl. f. Augenh. **79**:345-351, 1927.

ANTAGONISTIC DRUGS

The resultant effect on the pupil and on accommodation of any two drugs acting in opposite directions is purely quantitative. Thus the action of a drug of low potency (such as euphthalmine) may be greater, if instilled in sufficiently high concentration, than the action of a very potent antagonist (such as physostigmine) instilled in extremely dilute solution. Also, the length of action of a drug is important in that the effects of one drug (such as physostigmine) may predominate for a while and then the effects of the other drug (such as atropine) become apparent. If two highly potent antagonists are administered, the result may differ materially in different persons, and the pupil and accommodation will remain in an unstable state (not responding to physiologic stimuli in a predictable manner) while both drugs are acting. This is well exemplified by the unstable accommodative ability which results when physostigmine is instilled in a homatropinized eye.

In general, the potency of physostigmine is approximately equal to that of atropine, less than that of scopolamine and greater than that of homatropine (in equal concentrations). Physostigmine in a 0.25 to 1 per cent solution will always partially or completely counteract a mydriasis brought about by a strong preparation of epinephrine or one of its derivatives, while a maximal miosis produced by physostigmine is usually only slightly reduced by epinephrine. (The statement of Gautier [60] that physostigmine will not diminish mydriasis produced by epinephrine while epinephrine completely overcomes miosis produced by physostigmine has been widely quoted and misinterpreted: Actually, Gautier's experiments were performed on frogs, and the drugs were administered by injection into the dorsal lymphatic sac!) Prostigmine possesses a potency of the same order of magnitude as that of physostigmine. Pilocarpine is much less active than physostigmine, its miotic effect being easily overcome by most drugs of the atropine series and being approximately equivalent to the mydriatic effect of the epinephrine series. Carbaminoylcholine is about as active as pilocarpine, and acetylcholine and acetylbetamethylcholine are much less effective on instillation; however, either of the latter drugs may overcome even mydriasis due to atropine for a short time if injected subconjunctivally. The ability of histamine in a 10 per cent solution to produce a maximal miosis even in a fully atropinized eye has already been mentioned.

SYNERGISTIC DRUGS

If two drugs administered in physiologically equivalent concentrations produce a greater effect than twice that amount of either drug

60. Gautier, C.: Actions successives de l'esérine et de l'adrénaline sur la pupille de l'oeil de grenouille in vivo, Compt. rend. Soc. de biol. **87**:1402-1403, 1922.

alone, these drugs are known as synergists. From the standpoints of the pupil and accommodation, synergism may be accomplished in two definite ways: 1. By one drug acting to enhance the effect of the other. For example, physostigmine or prostigmine greatly increase the miotic action of acetylcholine or of acetylbetamethylcholine (by preventing their rapid destruction). Cocaine markedly increases the mydriatic response to epinephrine or its derivatives. 2. By both drugs accomplishing the same result while affecting different structures. For example, maximal dilatation of the pupil is accomplished by atropine (marked paresis of the sphincter) and by epinephrine (contraction of the dilator as well as some paresis of the sphincter). Also, a synergistic effect may sometimes be obtained with two drugs which accomplish the same result by acting on the same structure but in a different manner; an example is the action of any commonly used "miotic" plus the tonus increasing action of barium chloride. Synergism of this last type possibly takes place in the combined action of an atropine derivative with an epinephrine derivative in producing cycloplegia.

From a negative standpoint, synergism is never accomplished with two drugs which act in an exactly similar manner. Thus acetylbetamethylcholine is not a synergist of carbaminoylcholine or of pilocarpine; atropine is not a synergist of homatropine, and epinephrine is not a synergist of neo-synephrin. Physostigmine is not a synergist of pilocarpine [39] because physostigmine (which acts through the protection of acetylcholine and thus has the same point of action as pilocarpine) does not prevent the destruction of pilocarpine. Also, synergism of the first type mentioned is usually more marked with low concentrations of drugs than high concentrations, simply because the contraction of an intraocular muscle is subject to a certain maximal limit; for example, a choline compound probably adds nothing to miosis induced by a 10 per cent solution of histamine, because the maximal response of the sphincter is already attained.

From a practical standpoint, synergistic actions on the pupil and accommodation are of importance in three instances:

1. For breaking posterior synechiae in iritis, a combination of an atropine compound, an epinephrine compound and of cocaine is of theoretic value. Various combinations of these drugs are extensively used for this purpose and require no further elucidation.

2. In obtaining effective cycloplegia of short duration for refraction, considerable attention has been paid to a combination of benzedrine or paredrine with homatropine or atropine. While the theoretic efficacy of such a combination is not to be denied, the sum total of recent investigations along this line appears to be merely the proof that effective cycloplegia for refraction may be obtained by careful instillation of

much smaller quantities of homatropine or atropine than are generally employed and that such cycloplegia is consequently of shorter duration.

Myerson and Thau [25] stated that a synergistic effect on accommodation was obtained with dilute solutions of atropine and of benzedrine. Beach and McAdams [61] found that a single instillation of a 1 per cent solution of atropine plus an instillation of a 1 per cent solution of benzedrine produced almost as marked relaxation of accommodation as multiple instillations of a 1 per cent solution of atropine alone and noted the same thing with regard to a 5 per cent solution of homatropine plus a 1 per cent solution of benzedrine as compared with multiple instillations of a 2 per cent solution of homatropine alone. Since a shorter period of cycloplegia results after a single instillation of atropine or homatropine than after multiple instillations, they advised a combination of 1 or 2 drops of atropine or homatropine plus 1 drop of a 1 per cent solution of benzedrine for routine refractions. Powell [62] arrived at much the same conclusions. Tassman,[53b] substituting a 1 per cent solution of paredrine for benzedrine, found that a combination with 1 drop of a 4 or 5 per cent solution of homatropine or a 1 per cent solution of atropine similarly produced adequate cycloplegia, and Marron [63] also found a combination of homatropine and paredrine effective. Weinman and Fralick obtained good cycloplegia with either benzedrine or paredrine plus homatropine. Sudranski,[45a] however, found that 1 drop of a 5 per cent solution of homatropine alone produced almost complete cycloplegia, which was not enhanced by the addition of a 1 per cent solution of benzedrine, and Thorne and Murphey [45b] demonstrated that multiple instillations of a 2 per cent solution of homatropine alone, one instillation of a 5 per cent solution of homatropine alone or one instillation of a 5 per cent solution of homatropine plus a drop of a 1 per cent solution of benzedrine or of paredrine all resulted in essentially the same degree of cycloplegia. It is possible that a combination of stronger solutions of benzedrine or paredrine than have been used (or of the still more potent neo-synephrin) with weaker solutions of homatropine may prove of some advantage. Cocaine has been used to enhance the cycloplegic effect of homatropine by certain ophthalmologists for a great many years, but cocaine often disturbs the transparency of the corneal epithelium.

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61. Beach, S. J., and McAdams, W. R.: (*a*) Benzedrin in Refraction: Preliminary Report, Tr. Am. Ophth. Soc. **35**:221-226, 1937; (*b*) Benzedrin in Cycloplegia, Am. J. Ophth. **21**:121-124, 1938.

62. Powell, L. S.: The Practical Use of Homatropine-Benzedrine Cycloplegia, Am. J. Ophth. **22**:956-959, 1939.

63. Marron, J.: Cycloplegia and Mydriasis by Use of Atropine, Scopolamine, and Homatropine-Paredrine, Arch. Ophth. **23**:340-350 (Feb.) 1940.

3. In attaining maximal miosis (and other poorly understood effects) for reduction of tension in acute glaucoma, a combination of physostigmine or prostigmine with a choline compound is of theoretic value. The investigations of Clarke [43] using prostigmine and acetylbetamethylcholine suggest that the combination is of real practical value. It must be remembered, however, that maximal miosis and possibly also maximal effects on other intraocular structures are obtained with large doses of prostigmine or physostigmine alone and that in such a case the addition of acetylbetamethylcholine may not increase the effect.

A summary of the qualitative effects of the more commonly used "miotics" and "mydriatics" on the individual intraocular muscles is given in the accompanying table. The mode of action of these drugs and their quantitative effects cannot well be summarized in this manner.

Qualitative Effects of Various Drugs on the Intraocular Muscles *

Drug	Effect on Iris Dilator	Effect on Iris Sphincter	Effect on Ciliary Muscle
Acetylcholine	0	+	+
Acetylbetamethylcholine (mecholyl)	0	+	+
Carbaminoylcholine (doryl)	0	+	+
Pilocarpine hydrochloride	0	+	+
Physostigmine salicylate	0	+	+
Prostigmine bromide	0	+	+
Atropine sulfate	0	−	−
Homatropine hydrobromide	0	−	−
Scopolamine hydrobromide	0	−	−
Atropine methylnitrate	0	−	−
Duboisine sulfate (hyoscyamine)	0	−	−
Euphthalmine hydrochloride	0	−	−
Epinephrine hydrochloride (laevo-glaucosan)	+	−	−
Neo-synephrin hydrochloride	+	−	− (?)
Benzedrine sulfate	+	−	− (?)
Paredrine hydrobromide	+	−	− (?)
Ephedrine sulfate	+	−	−
Cocaine	+	−	−
Histamine (amino-glaucosan)	+ (?)	+	(?)
Ergotamine tartrate	+	+	(?)

* Increase in tonus is indicated by +, decrease in tonus by − and no effect (in therapeutic concentrations) by 0.

News and Notes

EDITED BY W. L. BENEDICT

UNIVERSITY NEWS

Dedication of Library and Auditorium.—The University of Oregon Medical School dedicated its new library and auditorium on June 7, 1940. A large bronze bas-relief of Dr. John E. Weeks in profile, the work of the eminent sculptor Gabriel Lavare, was presented by the many friends of Dr. Weeks. Beneath it is the following inscription:

JOHN E. WEEKS, M.D., Sc.D. LL.D.

WHOSE GENEROUS DONATION MADE POSSIBLE THIS BUILDING

PIONEER IN RESEARCH

RESTORER OF SIGHT

TEACHER OF TEACHERS

"LABOR OMNIA VINCIT"

GENERAL NEWS

Graduate Course in Ophthalmology and Otolaryngology.—The fifteenth annual spring graduate course in ophthalmology and otolaryngology will be given at the Gill Memorial Eye, Ear and Throat Hospital in Roanoke, Va., April 7 to 12, 1941. The following guest speakers will appear on the program: Dr. LeRoy A. Schall, Boston; Dr. Alfred Cowan, Philadelphia; Dr. Robert Groff, Philadelphia; Dr. J. H. Maxwell, Ann Arbor, Mich.; Dr. Paul Moore, Cleveland; Dr. A. D. Ruedemann, Cleveland; Dr. C. R. Straatsma, New York; Dr. A. B. Reese, New York; Dr. Raymond Meek, New York; Dr. Warren Vaughan, Richmond, Va.; Dr. Stewart Nash, Rochester, N. Y., and Dr. Frank Walsh, Baltimore.

FOREIGN NEWS

Prize Offered for Essay in Ophthalmology.—The Ophthalmological Society of the United Kingdom announces the Treacher Collins Prize, which is awarded triennially for the best essay on a subject chosen by the council of the society. The prize is open to qualified medical practitioners of any nationality but must be written in English. The subject is "Allergy of the Eye and Its Adnexa." Essays should be submitted before December 31 to the honorary secretary, Ophthalmological Society of the United Kingdom, 5 Racquet Court, Fleet Street, London, E. C. 4. The name should not be on the essay; rather, a distinguishing pseudonym or quotation is to be used, which should also be on a sealed envelope containing the candidate's name and address to accompany the essay.

Obituaries

SIR GEORGE A. BERRY, M.D., LL.D., F.R.C.S.
1853-1940

Sir George Andreas Berry died on June 18, at his residence at North Berwick, Scotland, at the age of 87 years. He was born in Glenstriven, Argyllshire, Scotland, in 1853. During his preliminary education he devoted himself to mathematics and graduated in medicine in 1876. Berry received his introduction into ophthalmology in a number of Continental hospitals and universities, particularly in Copenhagen, Denmark, where he studied with his distinguished uncle, Prof. Hansen Grut. He was resident at the Royal London Ophthalmic Hospital (Moorfields) in 1878-1879.

Berry's textbook on diseases of the eye was most popular, in his own country as well as on the Continent and in America. This book was referred to by many leading Continental professors of ophthalmology as being not only an exhaustive treatise but distinguished by many original observations and ideas. In addition, two monographs have been particularly outstanding; their titles are "Subjective Symptoms in Eye Disease" and "Ophthalmoscopic Diagnosis."

Berry was one of the moving spirits in the formation of the Ophthalmological Society of the United Kingdom in 1880, was a well known figure at the meetings and was president of this society in 1909-1911. In 1917 he delivered the Bowman Lecture on "Colour Sense Phenomena and Some Inferences Which They Seem to Suggest."

Berry was a member of the staff of the ophthalmic department of the Royal Infirmary, Edinburgh, Scotland, for twenty-three years and a lecturer on diseases of the eye in the Edinburgh University.

He was a member of many societies and received numerous honors, including that of being appointed honorary surgeon oculist in Scotland to King Edward VII and later to King George V. In 1916 knighthood was conferred on him. From 1922 to 1931 he was a member of parliament for the Scottish universities, and at the conclusion of this period he received the honorary degree of LL.D. from Edinburgh University.

Dr. A. H. H. Sinclair, who wrote the obituary in the British Medical Journal of June 29, 1940, from which the foregoing data have been taken, stated: "Sir George Berry was a man of remarkable ability. Physically vigorous, he enjoyed outdoor sports—especially golf and

angling. He was also much interested in music, and played the 'cello. . . . His great interest in mathematics was abundantly shown in his textbook and in his contributions to this subject. He possessed a remarkable acquaintance with modern languages, and especially with the less generally known Norse tongues. . . . His brightness of mind, scientific imagination, and high and varied attainments were fully recognized and highly valued by his colleagues, and he was much beloved by a wide circle of friends."

ARNOLD KNAPP.

Questions and Answers

CONICAL CORNEA

To the Editor.—I should like what information I can get on conical cornea. I have 3 or 4 cases that I have been following for the past ten years. The condition is progressive, and the patients require stronger minus cylinders at each refraction. I have used vitamins and drops of epinephrine hydrochloride.

D. W., M.D.

Answer.—While the mechanism of the formation of keratoconus is relatively clear, its cause remains a mystery. Keratoconus develops due to bulging of thinned cornea in an eye that has normal or even subnormal intraocular tension. The maximum bulging is near the center of the cornea, this region being farthest from the vascular limbus. Why the cornea should be weakened is unknown. Some ophthalmologists feel that there is a congenital defect. There are reports of more than one case in a family. Others think that the condition is due to an endocrine imbalance. The onset usually occurs at about the age of puberty, when the endocrine balance is changing. Enlargement of the sella turcica has been found. Changes from the normal in sugar metabolism and in excretion due to a lowered renal threshold have been demonstrated. Diet is considered the important factor by certain ophthalmologists, who have presented some convincing evidence that vitamins A and D play roles. At any rate, keratoconus rarely occurs in well nourished robust persons. It occurs in persons of inferior constitution, the avascular cornea being weakened along with a generalized inferiority of the tissue. As the cornea yields to pressure, the central part of the stroma may be thinned up to one fifth of its normal thickness. The cornea does not rupture unless complicated by ulcer from inadequate protection to the lid.

As in other conditions of obscure causation, many methods of treatment for keratoconus have been advocated, and none may be called satisfactory. Treatment may be divided into medical, refractive and surgical.

The general condition of the patient should be improved by the usual methods as indicated in the case at hand. Epinephrine hydrochloride instilled in the conjunctival sac may be useful. Whether this acts by nutritional changes to the cornea or by lowering of the intraocular tension is not clear. In some cases it seems to stop the progression. Recent reports of arrest by injections of anterior pituitary extract and of extract of adrenal cortex suggest that these may be of use. Large doses of vitamins A and D may be of value. Their effect may be specific, and if not their general tonic effect may help. Miotics may improve vision by the optical effect of a small pupil. Pressure bandages worn at night have been used to relieve the tension on the cornea.

The refraction should be checked frequently and the glasses changed as often as necessary to keep the visual acuity at its maximum. Refraction is best done with a small pupil or a pinhole. The glasses should

be worn close to the eyes. If spectacles do not give useful vision, contact glasses may be fitted. This is not an easy task, and patience on the part of both physician and patient is necessary. These glasses not only markedly improve vision but tend to arrest the progression of the process, perhaps due to a counter pressure on the anterior corneal surface.

If useful vision cannot be maintained under the foregoing treatment, surgical intervention is indicated. It cannot be expected to yield brilliant results. Cautery, physical or chemical, to the apex, paracentesis, optical iridectomy, discission, cataract extraction and excision of an elliptic flap of cornea above the pupil or directly over the pupil following the method of Shimlsin have all been tried, as have other procedures, with varying degrees of success and failure.

CATARACT EXTRACTION AND HIGH TENSION

To the Editor.—A patient of mine, a Negress aged 30, has bilateral congenital central nuclear cataract. She has peripheral vision that enables her to get around and take care of herself, although she can only count fingers at 10 feet (3 meters). Complicating her cataracts is a tension of 25 mm. of mercury (Schiötz tonometer) that I am barely able to keep down to 22 mm. with a 2 per cent solution of pilocarpine hydrochloride. What shall I do for this patient? I am afraid if I merely do an iridectomy to relieve the tension and then extract the cataract in a few weeks that the tension will rise again to where it was before I get around to the extraction. On the other hand, a fistula type of operation (e. g., Elliot's trephine operation) will complicate the cataract extraction. Which procedure would you advise? Please give me in detail the exact technic for cataract extraction performed after an Elliot trephine operation. T. O. G., M.D.

Answer.—Intraocular tension of 25 mm. of mercury (Schiötz) is not inconsistent with normal visual acuity and may be within normal limits of variation of tension for the person concerned. Without other evidence of impairment of ocular function, such as contraction of the visual field or scotomas about the blindspot, and in the absence of cupping of the optic disk, tension of 25 mm. (Schiötz) is not sufficient reason for surgical intervention. Instillation of miotics in eyes with nearly normal tension does not surely reduce the tension to figures below the normal variation—18 to 22 mm. (Schiötz). On the basis of the findings presented, a diagnosis of glaucoma is not substantiated and surgical intervention for lowering the intraocular tension would not seem to be indicated.

An operation for extraction of cataract can be made to serve the purpose of maintaining a low intraocular tension by employing a combined operation with a deep, broad iridectomy. It is true that glaucoma sometimes follows combined extraction of a cataract and that surgical procedures are required to lower the tension, but such a condition is not predictable. It does not predominantly follow any particular type of cataract operation and may be dealt with by cyclodialysis or an iris inclusion type of operation. Corneoscleral trephining after cataract extraction is likely to be complicated by the scar of the former incision

at the limbus. On the other hand, cataract extraction following trephining at the sclerocorneal limbus is not difficult. The incision may be made within the cornea anterior to the trephine opening so that the fistula is not disturbed. Extraction of the lens may then be accomplished by whatever method the operator may choose.

CHRONIC GLAUCOMA

To the Editor.—I have a patient who has chronic glaucoma in the left eye, with partial loss of vision. The right eye has normal fields and vision, and at times there is a slight increase in tension.

In such a case is surgical intervention on the right eye advisable or should one wait until it begins to show field or visual disturbances?

O. J. M., M.D.

Answer.—It is considered good practice to defer operation for glaucoma until signs of visual deterioration appear or until it can be definitely demonstrated that moderate use of miotics will not maintain the intraocular tension at a nearly normal level. To be sure, one cannot be certain that the tension remains continuously at a safe level, but the eye may be able to tolerate a tension somewhat above figures regarded as normal for some time without showing loss of function.

Operation on the left eye is indicated, but operation on the right eye should be deferred until it begins to show field or visual disturbances.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

OBSERVATIONS ON THE RATE OF SEDIMENTATION OF THE BLOOD CORPUSCLES BEFORE AND AFTER OCULAR OPERATIONS. R. KLAR, Klin. Monatsbl. f. Augenh. **102:** 321 (March) 1939.

Klar gives a synopsis of the test of sedimentation of the blood corpuscles, which was known to Hippocrates. He renders an account of his observations on 100 patients for whom this test was done forty-eight hours prior to bulbar and other ocular operations and twenty-four or seventy-two hours, respectively, afterward. Acceleration of sedimentation did not take place in any case in which operation was done within the eyeball or on its adnexa. In Klar's opinion the reason for this is the smallness of the wound in ocular operations.

K. L. STOLL.

Biochemistry

ABSORPTION OF DEXTROSE AND ASCORBIC ACID BY THE LENS. H. K. MÜLLER, Arch. f. Ophth. **140:** 258, 1939.

Verzàr and his associates discovered that dextrose during its passage from the lumen of the intestines into the blood stream is esterized with phosphoric acid and that the absorption of dextrose is largely dependent on this esterization. If the esterization is prevented by the presence of monoiodoacetic acid, the absorption of dextrose is inhibited. In the paper under review, Müller shows that the absorption of dextrose and of ascorbic acid by surviving lenses of pigs or rabbits is also inhibited by monoiodoacetic acid. Since this substance does not alter the permeability of the lens capsule for sodium thiosulfate, it is probable that the absorption of dextrose and ascorbic acid by the lens, just like the absorption of dextrose from the intestines, entails a process of esterization.

P. C. KRONFELD.

Conjunctiva

TREATMENT OF GONORRHEAL OPHTHALMIA. A. G. BOWER and W. FRANK, Am. J. Ophth. **22:** 277 (March) 1939.

Bower and Frank reach the following conclusions after a study of 21 cases of bacteriologically proved gonorrheal ophthalmia:

"(1) Sulfanilamide is of great value in the treatment of gonorrheal ophthalmia when combined with adequate local therapy.

"(2) Best results are obtained when a high blood sulfanilamide concentration is achieved early in the treatment and maintained long enough to secure negative eye smears.

"(3) Relapse can best be prevented by continuing sulfanilamide in small doses for 7 to 10 days after the first negative eye smear."

W. S. Reese.

Larval Conjunctivitis. H. R. Sniderman, Am. J. Ophth. **22**: 1253 (Nov.) 1939.

Sniderman reports the case of a 42 year old man who felt something hit his left eye. He was seen five hours later complaining of burning, lacrimation and something in his eye. At first nothing was found, but with a binocular loupe small wormlike organisms were seen in the lower fornix. Attempts to wash these out were unsuccessful, so the eye was anesthetized and 15 of the organisms hooked into the conjunctiva were removed with a cotton applicator. They were 1 mm. long and proved to be Oestrus ovis, the larva of the common sheep gadfly.

W. S. Reese.

Streptococcic Pseudomembranous Conjunctivitis Treated with Sulfanilamide. K. C. Swan and J. H. Allen, Am. J. Ophth. **22**: 1255 (Nov.) 1939.

Three patients with streptococcic pseudomembranous conjunctivitis of a serious nature were treated with sulfanilamide. The condition had progressed despite other forms of treatment. One patient had lost an eye in a previous attack. Signs of clinical improvement were evident after two or three days of treatment with sulfanilamide, and in 1 case a recurrence was controlled by further use of the drug.

W. S. Reese.

Comparative Ophthalmology

Ascorbic Acid Content of Eyes of Various Species. R. Vladesco and H. Stefanesco, Compt. rend. Soc. de biol. **132**: 169, 1939.

The authors' data for the ascorbic acid content of the aqueous, in milligrams per hundred cubic centimeters, are as follows: calf, 11; heifer, 15; ox, 14.5; lamb, 16.7; ram, 8.7; pig, 14; horse, 26; horse (cataract in eye), 2.8; dog, 6, and man, 5.

J. E. Lebensohn.

Congenital Anomalies

Macular Coloboma: Histological Report. A. Sorsby and J. O. Oliver, Brit. J. Ophth. **23**: 724 (Nov.) 1939.

The literature on macular coloboma contains 6 histologic reports. With the exception of 1 case, that of Rintelen reported in abstract, none is free from objection, and in none, barring the doubtful exception of Deyl's case and the inadmissible case of Janku, was the condition observed clinically.

The case reported by Sorsby and Oliver was that of a man aged 52, dying from cerebral thrombosis. The right eye was emmetropic and normal; the left was myopic to about 6 diopters and showed a typical nonpigmented macular coloboma excavated to at least 20 diopters.

The only myopic change was a small temporal crescent. The eye was excised. There was a distinct bulging of the sclera in the macular region, clearly separated by a normally shaped sclera extending to the disk.

Of the coats in the region of the coloboma, the choroid showed the most marked and the retina the least marked changes. Scleral changes, while prominent, did not compare with those seen in the choroid.

In the area corresponding with the region of greatest bulging the choroid was represented by a thinned stratum of connective tissue devoid of blood vessels and showing pigment in small amounts only. This avascular zone seemed to be limited to a small area, measuring some 3 to 4 mm. only. Elsewhere in the choroid the blood vessels were few in number but extremely large and distorted, appearing as flattened channels rather than tubular structures; the choriocapillaris was almost entirely deficient throughout. Pigment was deficient and irregularly distributed. The retina showed changes only from distortion of the globe and the condition of the optic nerve. The sclera almost throughout, but most marked posteriorly, was involved in a generalized ectasia; the fixed cells were virtually absent. The whole structure appeared almost nonnucleated. There was only a small amount of elastic tissue, for the most part made up of collagenous fibrils.

The optic nerve showed a complete lack of fibers, degeneration being complete. The changes were similar to those in typical colobomas but showed also a completely avascular zone corresponding to the macular region. The retina was less affected than it would be with so extensive a choroidal coloboma.

The authors believe the explanation of Treacher Collins concerning the origin of macular coloboma to be the least unsatisfactory.

W. ZENTMAYER.

Cornea and Sclera

CORNEAL ULCER AND SULFANILAMIDE. ROGGENKÄMPER, Klin. Monatsbl. f. Augenh. **103**: 211 (Aug.) 1939.

Roggenkämper says that for a period of six months he treated all persons with corneal ulcer with sulfanilamide. The substance was given internally and by conjunctival injection, but the author did not neglect to employ also the former therapeutic measures, such as shock therapy in the form of intramuscular injections of milk and the customary local methods. Thus the subconjunctival injection of sulfanilamide was an auxiliary measure in the 26 cases on which this report is based. In 15 of the 26 cases there existed post-traumatic serpiginous ulcers of various ages and extension, and in nearly all of them hypopyon ulcer existed. In all these cases the therapeutic results were surprisingly favorable as regards rapidity of cure as well as the final results.

Among these cases of serpiginous corneal ulcers, there were some which at the time of hospitalization gave the impression that only splitting of the cornea would prevent a panophthalmia. However, treatment with sulfanilamide made this unnecessary. The delicacy of the resulting corneal scars and the visual acuity were surprising. This applies to new cases in which the ulcers yielded to from one to three injections as well as to old cases in which nearly all hope had been

abandoned and in which from six to eight injections of 0.5 cc. each were necessary to produce epithelization. Surgical operations were unnecessary, and the author regards this as an essential advantage of sulfanilamide therapy. For the scrofulous and catarrhal ulcers of the cornea the effect of the subconjunctival injections of sulfanilamide was not as favorable as for the typical serpiginous traumatic ulcers, but even there a favorable effect could be noticed. It is possible that the bacterial superinfection of those ulcers was excluded by the sulfanilamide. Favorable effects of subconjunctival injections of sulfanilamide were observed also in 2 cases of persistent parenchymal infiltrations following erosions and in 3 cases of infected erosions. In superficial keratitis, however, no effect was noticeable. The author concludes that sulfanilamide is of great value in the treatment of infectious corneal diseases, and that it may make surgical operations superfluous for serpiginous ulcers.

J. A. M. A. (ZENTMAYER).

Experimental Pathology

EXPERIMENTAL STUDY ON THE ACTION PRODUCED BY ASEPTIC FOREIGN BODIES IN THE EYE. P. VEIL and DESVIGNES, Ann d'ocul. **176:** 577, 1939.

The authors give in considerable detail the results of their experiments with rabbits in which foreign bodies were inserted in the anterior chamber, in the iris, in the ciliary body and in the vitreous. The majority of the foreign bodies were tolerated by the eye fairly well, the exceptions being mercury, copper and nickle.

The eye of the rabbit does not behave entirely under these circumstances like the human eye (for example, detachment of the retina is easy to provoke in the rabbit). Considering the chemical nature of the foreign body, the experimental and clinical results were nearly identical. However, chemical intolerance is sometimes retarded.

The foreign bodies introduced experimentally into the iris and ciliary body were usually well tolerated, while clinically one often observed a rapid intolerance (iridocyclitis). The size and form of the foreign body play important parts in the tolerance of the foreign body. Also it is exceptional in the clinical cases for the foreign body to be aseptic. The result of the experiments here cited deals entirely with the tolerance of an aseptic foreign body present in the eye for a relatively short period. A bibliography accompanies the article.

S. H. MCKEE.

EFFECT ON THE RABBIT LENS OF DIVIDED AND PROTRACTED DOSES OF ROENTGEN RAYS. W. ROHRSCHNEIDER and R. GLAUNER, Arch. f. Ophth. **140:** 700, 1939.

Ordinarily, for high voltage roentgen therapy from 20 to 40 roentgens are given per minute. By moving the tube farther away from the patient and by using thicker filters, the flow of radiating energy can be reduced to 4 to 6 roentgens per minute, which procedure is called protracted roentgen therapy. It has given much better results in cases of malignant

growths in the esophagus, larynx or trachea than are obtained with the ordinary technic. The same dose of roentgen rays can thus be applied in three forms, namely, in one single dose (S.D.); in divided, or fractional, doses (F.D.), and in fractional protracted doses (F.P.D.). The effects of these three forms of roentgen therapy were studied on the eyes of rabbits, and it was found that 1,000 roentgens given in a single dose or in fractional protracted doses always causes permanent changes in the lens. The skin of the lids behaves differently as the minimum single epilation dose is four to six times smaller than the same dose given as fractional doses or as fractional protracted doses. This difference between the skin and the lens can also be expressed by saying that the cumulative effect of repeated doses of roentgen rays on the lens is much greater than on the skin. But even in the case of the lens, fractional and fractional protracted doses are somewhat less harmful than single doses. The latent periods are longer and the final lenticular opacities are less marked after fractional and fractional protracted doses.

P. C. KRONFELD.

PATHOGENESIS OF GALACTOSE CATARACT. A. BAKKER, Arch. f. Ophth. **140:** 531, 1939.

Surviving lenses develop fine cataractous changes if galactose is added to their nutritive fluid. Compared to galactose cataract in vivo, the opacities occurring in explanted lenses are insignificant. In addition to the local toxic effect of galactose, a general metabolic disturbance caused by the ingestion of galactose must be responsible for the development of galactose cataract. As a manifestation of this general metabolic disturbance, the author reports low serum calcium values in rats which had been fed galactose.

P. C. KRONFELD.

General Diseases

CORRELATION BETWEEN PRESSURE IN THE RETINAL ARTERIES AND GENERAL BLOOD PRESSURE IN DIABETES: REPORT OF CASES. A. FRANCESCHETTI and E. B. STREIFF, Klin. Monatsbl. f. Augenh. **102:** 633 (May) 1939.

The authors report the result of their research on the correlation between the pressure in the retinal arteries and the general blood pressure in cases of diabetes. Examination of 82 diabetic patients, ranging in age between 42 and 78 years, showed hemorrhages or foci in the retina in 31, or 38 per cent. Taking a diastolic pressure of 70 to 85 mm. of mercury as normal, they found these values in 50 patients (61 per cent). Hence, diabetic changes in the retina may occur without general hypertension. The supposition is unfounded that isolated retinal hypertension may cause diabetic retinitis in the presence of normal general blood pressure. Tables show the frequency of various findings on which the authors base their conclusion. The fact, surmised for a long time, is proved that in simultaneous occurrence of general hypertension and diabetes the latter is responsible for retinal changes.

K. L. STOLL.

OCULAR SYMPTOMS IN LEIODYSTONIA AND SPRUE. P. H. POCK-STEEN, Geneesk. tijdschr. v. Nederl.-Indië **79:** 1986 (Aug. 8) 1939.

Pock-Steen calls attention to the ocular symptoms that he observed in about 100 patients with leiodystonia, sprue and spruelike disorders. The most noteworthy ocular symptom was a reduced visual acuity during weak daylight, such as twilight, and during inadequate artificial illumination. The author designates this symptom as twilight blindness, or aknephascopia. Twilight blindness is due to a diminished susceptibility to the light waves. It differs in nature from the well known night blindness. It is not influenced by the administration of vitamin A, as is night blindness, but it is greatly amerliorated a few hours after the administration of approximately 1 mg. of riboflavin (vitamin B_2). Of 109 patients, 67 stated without hesitation that their vision was greatly improved after the first injection of 1 mg. of riboflavin, whereas 31 were able to corroborate this only after a longer period. The remaining 11 patients observed only a slight improvement in their visual acuity. The author observed that the symptoms of twilight blindness were usually greater in patients with sprue who had anomalies of refraction. Nevertheless, even for these patients surprisingly favorable results were obtained. In this connection the author mentioned a patient aged 64, who stated that in two years he was obliged to change his glasses three times for stronger lenses, but that after his sprue had been treated with injections of riboflavin he could return to the use of his first pair of glasses. The author thinks that twilight blindness is a symptom of a general B_2 hypovitaminosis and the only one known that can be observed clinically. Other ocular symptoms observed in cases of sprue are mydriasis, conjunctival irritation, keratitis and disturbances of accommodation. The burning pains of conjunctivitis are also found to decrease after the administration of riboflavin. The author thinks that the ocular symptoms of patients with sprue are partly the result of B_2 hypovitaminosis and partly the consequence of the histamine toxicosis prevalent in sprue.

J. A. M. A.

Glaucoma

FAMILIAL PRIMARY GLAUCOMA IN ADULTS. A. H. BRIGGS, Brit. J. Ophth. **23:** 649 (Oct.) 1939.

Briggs discusses the various theories advanced to explain familial glaucoma and reports 2 families in which glaucoma simplex appears to be a familial condition.

He gives the following summary and conclusions:

"A survey of the scanty literature shows that familial glaucoma—a condition distinct from buphthalmia—appears to be a definite entity, although comparatively rare. All forms of glaucoma may occur in affected families, but the chronic simple type seems to be the most frequent. The condition is transmitted by both sexes and may involve several generations. There is some tendency to 'anticipation' in succeeding generations. The sexes are about equally affected, males being slightly more commonly affected than females. The mechanism by which the heredity influence acts is unknown; definite development of the

angle of the anterior chamber seems to be the most probable means, but the statistics suggest that the determining causes of buphthalmia, heredity glaucoma, and nonheredity glaucoma are separate and distinct. There seems to be no association between hereditary primary glaucoma and buphthalmia. There is little evidence to support the suggestion that hereditary glaucoma is due to some degree of congenital microphthalmia with a disproportionately large lens.

"Two glaucomatous families are described. The first family was probably affected through three generations, but no cases have yet been discovered in the fourth, although curiously enough, several members of the fourth generation have defective sight; three which have come under observation, however, have been found to be suffering from quite unrelated eye disorders and to show no signs of glaucoma.

"Of the third generation, of eleven legitimate children, two died in infancy; of the remaining nine, three were certainly glaucomatous and two more suffered from eye defects which were very probably glaucoma; two more had defective sight which may have been due to glaucoma, and two were unaffected. One of the two unaffected members died at the age of 24—too young for her condition to be certain.

"One member of the family developed a spontaneous choroidal detachment about three months after sclero-corneal trephining, and this persisted unchanged for a year. It was (unsuccessfully) treated by a cautery puncture and is still present.

"The second family comprises six siblings, eight are said to be glaucomatous, and six have been examined. Of these five were glaucomatous, and one was unaffected."

W. ZENTMAYER.

DAILY VARIATIONS OF DARK ADAPTATION IN GLAUCOMA. I. PRZHIBYLSKAYA, Vestnik oftal. **14:** 37, 1939.

The purpose of this paper was to establish the existence of variations between the night and the morning dark-adaptation curves of glaucomatous patients. Sixty-five eyes with symptoms suggestive of glaucoma were examined, and 130 adaptations curves were obtained by the adaptometer, devised by the author. The examination was done for a period from thirty to forty minutes. In all cases the curve obtained in the evening was lower than the one obtained in the morning. The findings were considered positive if the variation in the former was at least half that of the latter. Three drawings and some case histories illustrate the article. This examination of the daily variations of dark-adaptation curves is a new method for early diagnosis of glaucoma, particularly in the absence of changes of the other functions of the eye and with good tension during the day. Most likely these variations reflect the variations in tension.

O. SITCHEVSKA.

RESULTS OF THE ELLIOT OPERATION ON TWO HUNDRED AND FORTY-FOUR GLAUCOMATOUS EYES. H. THOMSEN, Acta ophth. **17:** 301, 1939.

Sclerocorneal trephining, according to Elliot's technic, was performed on 157 eyes. Bentzen's modification was used on 87 eyes. In this pro-

cedure a previously undermined conjunctival flap is brought down and sutured to scarified cornea, thus covering the trephine opening. The author regards this operation as easier and safer than the original method. In 75 per cent of all the cases the increased tension was satisfactorily reduced. In glaucoma simplex a reduction was obtained in 91 per cent. Late infections occurred in 4 cases; in 2 of these the infection was probably endogenous. The postoperative period of observation of these patients was variable, from a few months to many years.

O. P. PERKINS.

Lens

THE INTRA-CAPSULAR EXTRACTION OF CATARACT WITH FORCEPS: IS ITS USE JUSTIFIABLE? R. BUXTON, Brit. J. Ophth. **23:** 505 (Aug.) 1939.

The question presented in the title of this article is considered under the following heads: (1) historical and general consideration; (2) certain complications—their incidence, causes and avoidance; (3) choice of cases, and (4) important details of technic. A summary and conclusions and a list of references complete the article, which is lengthy. It is replete with the opinions, experiences and methods of other authors regarding intracapsular extraction. The author's summary and conclusions follow:

"(1) The advantages and disadvantages of the intra-capsular extraction of cataract with forceps are discussed.

"(2) The incidence of certain complications of the operation is investigated.

"(3) The importance of a careful selection of cases and attention to minute details of technique is emphasised.

"(4) Not until further statistics are published, showing the end-results of selected cases compared with the end-results of the classical operation, can any conclusions be arrived at as to whether the use of intra-capsular operation is justified.

"(5) While possessing certain advantages, the intra-capsular operation carries with it an increased risk of certain complications.

"(6) The results of the classical operation are so satisfactory that it is doubtful whether it will be superseded by the intra-capsular operation even in selected cases.

"(7) It is probable that under poor conditions where increased risks have to be taken, e. g., in India, the intra-capsular operation in selected cases is preferable."

W. ZENTMAYER.

VITAMIN C METABOLISM OF PATIENTS WITH SENILE CATARACT. P. KARBACHER, Arch. f. Ophth. **139:** 748, 1939.

J. Seefried reports that patients with senile cataract require for saturation a greater intake of vitamin C than controls of the same age (*Arch. f. Ophth.* **138:** 620, 1938). Karbacher, of Vogt's clinic, finds no significant difference in saturability with vitamin C between patients with cataract and normal controls of the same age. It has, therefore,

not been shown with certainty that there is a systemic disturbance of vitamin C metabolism in patients with senile cataract.

P. C. KRONFELD.

Neurology

OPTIC NEURITIS WITH TRANSIENT BLINDNESS AND MENINGOENCEPHALITIS AFTER VACCINATION FOR SMALLPOX: REPORT OF A CASE. H. SCHEYHING, Klin. Monatsbl. f. Augenh. **102:** 223 (Feb.) 1939.

Two weeks after vaccination for smallpox, a child aged 14 months appeared to be suddenly blind and did not care to sit upright. The child was perfectly well one week after vaccination, but fever was observed two days later. Meningoencephalitis was found accompanied by the following symptoms: optic neuritis, edema of the retina, restlessness, fever, opisthotonos, increased pressure of the cerebrospinal fluid and an increased number of cells with a negative Pandy reaction. Bacteria were absent in the spinal fluid. Recovery was complete after five weeks. Contamination of the lymph used for vaccination and reactivation of a previous infection could be excluded. Scheyhing, therefore, is of the opinion that the disease was caused by the vaccine virus used. Other cases of postvaccinal encephalitis are cited, among them being 1 case in which blindness was permanent. The author refers to the greatly exaggerated danger of vaccination in the mind of certain lay circles and stresses the extreme scarcity of complications occurring in the immense number of vaccinations done.

K. L. STOLL.

Ocular Muscles

ON DOUBLE VISION AFTER SQUINT OPERATION. A. STERNBERG-RAAB, Brit. J. Ophth. **23:** 568 (Aug.) 1939.

Fusion may be prevented (1) by a postoperative abnormality in the position of the eyes; (2) by abnormal retinal correspondence, and (3) by a difference in the size and shape of the retinal images in the two eyes.

Double vision may be caused by squint due to the operation and in a direction contrary to the former one, i. e., divergence after convergence and vice versa, or it occasionally occurs when the operation has been insufficient. Postoperative double vision in such cases is in all probability connected with the fact that the onset of squint did not take place in earliest childhood and that amblyopia was not of too high a degree—vision not over (sic) 5/35. The issue of such postoperative double vision seems to depend on several factors, such as the strength of the fusion power and fusion amplitude, and further on the degree and direction of the squint. In those cases in which convergence faculty is deficient and cannot be developed, the patient will sooner or later rid himself of the discomfort of double vision by suppression, and divergence can be eliminated only by another operation. When suppression appears in persons incapable of convergence, reoperation is justified.

W. ZENTMAYER.

Operations

SUGGESTIONS CONCERNING THE TECHNIC OF KERATOPLASTY. M. VANNAS, Arch. f. Ophth. **139:** 709, 1939.

The experiences gained from 18 keratoplasties on man and numerous keratoplasties on rabbits are reported. Trephining from behind proved to be the best method for obtaining the graft from the donor's eye. Its anterior segment was severed from the posterior segment by a preequatorial incision. The contents were removed and the cornea put on and inverted over a convex support (the epithelium being in contact with the support). The trephining was done in this position of the cornea, and a round graft with the desired degree of beveling of the edge was obtained. According to the author, the advantage of this method is that the most delicate portion of the cornea, the endothelium, is cut cleanly and not torn, as so often happens during trephining in the usual direction from in front backward. During the removal of the leukomatous piece of cornea from the eye of the donee, the lens is protected by a spatula made of white beechwood, which is introduced into the anterior chamber through a small keratome incision. In the absence of anterior synechiae the trephining may be done without protecting the lens. A shallow anterior chamber can be deepened by injection of a 10 per cent solution of gelatin with a needle which has been sealed at the tip and has a hole a few millimeters back of the tip (Leber's cannula). Vannas fastens the graft with a rectangular piece of a thin rubber membrane, which is held in place by sutures inserted in the episclera. This rubber membrane, through which the graft can be watched, is removed on the twelfth day. The grafts were obtained either from the eyes of newborn infants who had died shortly after birth or from the eyes of adults which were enucleated in vivo. The eyes were kept in cod liver oil at a temperature of + 4 C. If the donor had been a newborn infant, the eye of the donee became myopic because of the short corneal radius. This myopia was corrected with contact glasses. P. C. KRONFELD.

Physiology

A STUDY OF DIET IN RELATION TO HEALTH: DARK ADAPTATION AS AN INDEX OF ADEQUATE VITAMIN A INTAKE. A. M. THOMSON, H. D. GRIFFITH, J. R. MUTCH, D. M. LUBBOCK, E. C. OWEN and G. LOGARAS, Brit. J. Ophth. **23:** 697 (Nov.) 1939.

This lengthy paper is replete with tables and graphs. The authors undertook the determination of normal standards of dark adaptation in children on different diets and the effects of administration of vitamin A in individual cases. As their method is possibly the only one in which white light was used throughout the study, in order to test whether negative results obtained might be due to the use of white light in the photometer a violet filter was used. From these results they concluded that aperture 5, with the violet light, was roughly equivalent to aperature 2 on the standard apparatus. The subjects of the rate of vitamin A in light perception, the rate of substances other than vitamin A in dark

adaptation, night blindness in pathologic conditions and clinical night blindness are discussed.

The authors reach the following conclusions:

"It would appear from our results that neither rate of dark adaptation nor light threshold of the fully dark adapted eye has necessarily a close correlation with intake of vitamin A in the diet. Considerable variation may occur in one individual and between individuals on the same diet.

"Clinical observations and experiments in vitamin A deficiency have shown that vitamin A and dark adaptation are related. But it seems that in certain cases at any rate the speed and extent of cure of hemeralopia by vitamin A concentrates are subject to considerable variation.

"The majority of workers believe that the study of dark adaptation can be used for a test for vitamin A deficiency, but until differences in technique and interpretation of results have been resolved, it is impossible to be certain how far their recorded observations represent physiological facts."

W. ZENTMAYER.

Refraction and Accommodation

A STUDY OF THE PROFESSIONS TESTING OCULAR REFRACTION. E. A. THACKER, Am. J. Ophth. **22:** 1227 (Nov.) 1939.

Thacker makes the following summary of this interesting study:

"A study of 1,416 students has revealed that 37.8 per cent examined by the optometrist and 19 per cent by the oculist did not have normal vision with glasses as determined by the Snellen test. After deducting those cases with marked change in visual acuity within the last year there still remained 32.3 per cent examined by the optometrist and 6.7 per cent by the oculist who had defective vision with glasses.

"After deducting those students with visual changes since their last examination by the oculist and optometrist there remained 1.9 per cent and 11.1 per cent, respectively, of the students with symptoms related to ocular disorders.

"Practically three fourths of the students were unable to differentiate the oculist from the optometrist.

"The average cost of the ocular examination and glasses was about 10 per cent greater in the oculist group than in the optometrist group.

"There is need for better medical regulations to determine the proficiency, and for the registration and licensing of physicians representing themselves as specialists.

"The Class-A schools of optometry have raised their requirements to a four year course. Several of these schools are attempting to teach pathology and diagnosis. Examples of the curricula are given. In the light of existing circumstances it would seem that much of the teaching is futile. Since a knowledge of general medicine is a necessary foundation for the study of the etiology, pathology and diagnosis of ocular disease, and there exists such a limited number of capably staffed postgraduate institutions, optometrists according to their present preparation can never serve the public as efficiently as can the ophthalmologists. Eventually, if the practice of optometry is to continue in the direction it is now pursuing, the medical schools may find it necessary to absorb the teaching and control the practice of this profession.

"Otherwise, when and if the professional standards and ethics for the practice of optometry reach a higher plane, it may be possible that the same cooperation may exist between the medical and optometry professions as is now present between the practices of medicine and dentistry in serving the public efficiently."

W. S. REESE.

Retina and Optic Nerve

CASE OF A GIANT HOLE OF RETINA. J. C. MARSHALL, Brit. J. Ophth. **23:** 369 (June) 1939.

A man aged 33 had been blind in the right eye for some years. There was complete detachment of the retina in the left eye. Extending from 12:30 to 6:30 o'clock there was a marked fold of the retina rolled inwardly from the margin of the tear. The horizontal width of the tear was 8 to 10 disk diameters; the base showed details with a —16 D. sph. The retina was seen in detail at the upper and outer quadrant with a —10 D. sph. The subretinal fluid was drained, and thirteen days later a perforating diathermy operation with Šafář needles was performed. One month later there was no improvement. Ten months later the tear was reduced. Two years later there was complete cataract with good projection of light. The cataract was removed, and a week later the details of the fundus could be seen. The tear was healed. Subsequently, capsulotomy was done. Several months later with + 8 D. sph. vision equaled 6/24; with + 11 D. sph. the patient could read Jaeger's test type no. 12.

The article is illustrated.

W. ZENTMAYER.

TUBERCULOSIS OF THE OPTIC NERVE AND PAPILLA. P. DIEFFENBACH, Ann. d'ocul. **176:** 497 (July) 1939.

Cases of tuberculosis of the optic nerve with anatomic proof are not numerous in the literature. For this reason the authors report in detail the case of a man of 23 who came to the clinic in January 1938, requesting examination of the eyes. His personal history revealed that he had had dry pleurisy at the age of 4 years as well as a disease of undetermined nature, which he believed was tetanus. He had been in the hospital in June 1937 with serofibrinous pleurisy, and in November he had noted a rapid decrease in vision. His condition grew rapidly worse after admission, death occurring a month later.

The eyes were removed, and the results of the pathologic examination are given in detail, after which the subject is discussed and the pathogenesis, diagnosis and treatment are given in considerable detail.

Two illustrations and a large bibliography accompany the article.

S. H. MCKEE.

A PECULIAR DISEASE OF THE MACULA. H. RIEGER, Arch. f. Ophth. **140:** 456, 1939.

Five cases of unilateral macular disease in young or middle-aged patients are reported. The typical picture was that of a yellowish white prominent retinal lesion, which was slightly smaller than the disk and

surrounded by deeply situated retinal hemorrhages. No lesions could be found elsewhere in the fundi. The disease was thought to represent a mild form of the external exudative retinitis of Coats. In one of Rieger's cases virulent tubercle bacilli could be grown from the patient's blood. The other 4 patients were definitely hypersensitive to tuberculin. All patients received tuberculin treatment. The final vision was good. The author considers tuberculosis as the "most important and most common" cause of the vascular changes which represent the primary changes in external exudative retinitis.

P. C. KRONFELD.

DEFICIENCY DISEASES OF THE OPTIC NERVE. A. HAGEDOORN, Arch. f. Ophth. **140:** 561, 1939.

A deficiency disease of the optic nerve may be caused by a deficient diet, by faulty absorption and utilization of the foodstuffs or by excessive metabolic consumption of vitamins. Chronic excessive ingestion of alcohol leads to a deficiency disease indirectly by causing gastric achylia, which entails faulty absorption. The pathogenesis of pernicious anemia and other hyperchromic anemias is discussed in the light of the work of Castle, according to which the normal reproduction of erythrocytes is dependent on the formation in the stomach and duodenum of a substance in which an extrinsic food-borne factor (vitamin B_2 or G) is combined with an intrinsic factor that is contributed by the pyloric and duodenal glands. Cases of retrobulbar neuritis combined with deficiency anemia are cited. Bilateral interstitial neuritis associated with diabetes is also interpreted as being caused by faulty absorption of food substances necessary for the integrity of the central nervous system.

P. C. KRONFELD.

Trachoma

THE EFFECT OF SULFANILAMIDE ON THE COURSE OF TRACHOMA. L. A. JULIANELLE, J. F. LANE and W. P. WHITTED, Am. J. Ophth. **22:** 1244 (Nov.) 1939.

This survey was carried out on Indians, with the exception of 6 white patients, the treatment recommended by Loe being adopted. The following conclusions were made:

"1. Sulfanilamide administered in trachoma as described caused varying degrees of improvement in about 40 per cent of the patients studied, and recovery in about 20 per cent, without affecting the remainder.

"2. The drug has a marked and rapid effect on the secondary infections commonly associated with trachoma.

"3. The most striking results were observed in patients with exacerbative disease.

"4. In a small number of patients studied (six), epithelial-cell inclusions disappeared during the course of treatment.

"5. Sulfanilamide induces a variety of toxic manifestations, which need not be dangerous if care and judgment are exercised."

W. S. REESE.

Tumors

LYMPHANGIOMA OF THE ORBIT. M. RADNOT, Arch. f. Ophth. **140**: 328, 1939.

Two cases of lymphangioma of the orbit are reported. Characteristic of these tumors, which are usually congenital, are repeated hemorrhages, as a reaction to which, in the course of time, lymphatic tissue develops.

P. C. KRONFELD.

TRAUMATIC ORIGIN OF SARCOMA OF THE CHOROID: REPORT OF CASES. W. KREIBIG, Klin. Monatsbl. f. Augenh. **102**: 333 (March) 1939.

Kreibig describes 7 cases of sarcoma of the eyes clinically and histologically. The tumors involved the lids and surrounding skin in 2 cases; intraocular sarcomas were present in 5 cases. The main purpose of this paper concerns the question of how far injuries may be responsible for this class of tumors. Irrefutable proof must be furnished of primary injury of the tissues with exclusion of preexisting pathologic changes at the site of the tumor. It must be considered, however, that the effect of trauma may manifest itself in a portion of the uveal tissue which is not injured. Again, any processes have to be taken into consideration which may produce formation of new cells and transformation of tissues. Proof in this respect will be easy in cases in which protracted inflammation sets in at once; it is practically impossible whenever the patient is seen for the first time with a shrunken eyeball. A long latent period suggests a causal connection between trauma and tumor more plausibly than a short period. Disposition to formation of such tumors may rest on the base of familial tendency or may be acquired during the course of the disease. Differentiation of these two types, theoretic at the present time, may be solved definitely later.

K. L. STOLL.

Visual Tracts and Fields

INFLUENCE OF OPACITIES OF THE MEDIA ON THE VISUAL FIELD. H. LAUBER, Arch. f. Ophth. **139**: 687 (Oct.) 1939.

The literature contains little information with regard to the influence of opaque media on the visual field. A priori one would expect that the isopter for a certain target is made narrower and a scotoma made larger by increasing lenticular opacities. Two cases are reported in which glaucomatous visual defects improved on removal of cataractous lenses. The evaluation of field defects in the presence of glaucoma plus cataract is difficult, but by close observation of the patient one may be able to tell whether or not these defects and lenticular opacities progress at the same rate. One of Lauber's assistants will report on extensive studies of this kind at a later date.

P. C. KRONFELD.

Therapeutics

SHORT WAVES IN OCULAR THERAPY. BIRCH-HIRSCHFELD, Klin. Monatsbl. f. Augenh. **102**: 107 (Jan.) 1939.

The author describes the methods of short wave therapy used for ocular diseases and the results obtained by other writers. This review

includes about 1,500 cases reported in 14 publications. Birch-Hirschfeld used an ultratherm constructed by Siemens, adapting the dosage to each individual case. A voltage of 19 applied for from ten to fifteen minutes was the average dose; he started with a period of exposure of five minutes in cases of painful disease and of involvement of the fundus. He reports on his results in 94 cases. Excellent results were obtained in 57 cases, good results in 24, slight improvement in 9 and no improvement in 4. In 3 of these 4 cases the patients suffered from glaucoma, while in the fourth the patient had parenchymatous keratitis. The best results were recorded for inflammatory and purulent conditions of the lids and tear sac, superficial keratitis and neuralgias in the realm of the trigeminus nerve. In 10 cases of superficial keratitis and in 2 cases of corneal ulcer ultraviolet rays were used simultaneously. Short wave irradiation effected prompt cure in 2 cases of spontaneous iritis; in 6 of 9 cases of tuberculous iridocyclitis a very good reaction was obtained, and in 3 other cases the response was fairly good. Prompt results were obtained in 3 of 4 cases of neuroparalytic keratitis, whereas extended treatment was necessary in the fourth case. Small doses of short wave radiation were used in cases of fundus disease, the fundus, the vision and the field of vision being under constant control. Marked improvement was noted in 2 of 3 cases of retinitis with hemorrhages, although fresh hemorrhages are likely to follow the treatment. Prompt and considerable improvement was noted in 3 of 5 cases of optic neuritis and in 15 cases of exudative chorioretinitis. No uniform results could be obtained in cases of opacities of the vitreous; in 3 cases the opacities cleared in a short time, in 3 they responded more slowly and in 3 they were not influenced.

Adverse results were not seen in any of the 94 cases. Short wave irradiation yielded good results, had an analgesic effect and shortened the time of treatment in cases of inflammatory and purulent disease of the lids, the lacrimal apparatus and the anterior segment of the eyeball. Satisfactory results were observed in cases of disease of the retina, choroid and optic nerve.

K. L. STOLL.

Society Transactions

EDITED BY W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

DAVID WEBSTER, M.D., *Chairman*

ROBERT K. LAMBERT, M.D., *Secretary*

April 15, 1940

Subacute Dacryoadenitis with Conjunctivitis Due to Fusiform Bacilli. DR. WEBB P. CHAMBERLIN (by invitation).

Fusospirochetal disease is a mixed infection in which are present fusiform bacilli, spirochetes and actively motile vibrios, usually associated with streptococci or staphylococci.

This group of organisms together produce the infections of the mucous membrane first described by Vincent. While various organs of the body may be attacked, the infection is most commonly seen as the foul, ulcerative lesions of Vincent's angina in the mouth and throat.

Fortunately, involvement of the eye is infrequent, and there are all told only 12 cases reported in the literature.

Bertozzi in 1907 reported a case of metastatic ophthalmitis due to fusiform bacilli. Since then there have been recorded 2 instances of dacryocystitis, 2 of orbital cellulitis secondary to sinusitis and 2 of localized ulceration of the conjunctiva or the margin of the lids.

The most frequent ocular lesion is a unilateral purulent conjunctivitis which heals without corneal involvement. There have been 6 such cases reported, among which the best analysis is found in the paper of Dunnington and Khorazo, of New York. They successfully treated their patients with a 1 per cent solution of sodium perborate. Herrenschwand reported a cure with the use of a solution resulting from the action of iodine on sodium carbonate solution (Pregl's solution), and Dejean and Temple found a 1.5 per cent solution of zinc sulfate efficacious. Recently, Dr. Macnie found an ointment containing mercury bichloride (1 : 3,000) satisfactory in treating his patient.

Gaudie and Sutherland have reported a single case of bilateral purulent conjunctivitis with extensive secondary corneal ulceration, loss of vision resulting due to dense leukomas in each eye. This is the only previously reported instance of bilateral conjunctivitis or secondary corneal involvement.

The following case is reported from the service of Dr. Brittain Payne at the New York Eye and Ear Infirmary.

A 39 year old Italian woman was first seen in November 1937, complaining of itching and discomfort of the eye for eighteen months and bilateral purulent discharge for two months. Examination revealed only the purulent conjunctivitis, with greatest involvement in the right eye.

Examination of a smear of the pus revealed a mixed bacterial flora, including gram-positive cocci, fusiform bacilli and spirilli. No anaerobic cultures were taken at that time. The patient received instillations of a number of antiseptics, with improvement, although the discharge never completely disappeared from the right eye.

During the succeeding year there were increasingly severe recurrences of the purulent conjunctivitis, particularly in the right eye, with the patient complaining of pain in the outer portion of the upper lid. No foci of infection were found, and serologic studies gave negative results. Despite energetic local and systemic treatment, a deep keratitis and iritis developed in the right eye. Because the eye was blind and painful, enucleation was performed in January 1939.

Pathologic study of this globe by Dr. Bernard Samuels revealed a much thickened cornea with deep vascularization. There was marked lymphocytic infiltration of the cornea, conjunctiva and episclera, but inflammation of the uveal tract was confined to the iris.

During the past few months the chronic purulent discharge from the remaining left eye has become more pronounced, and the patient has complained particularly of pain and tenderness referred to the outer portion of the upper lid.

Examination one month ago at the time of the patient's admission to the infirmary showed no inflammation of the right conjunctival cul-de-sac, though there was slight tenderness in the vicinity of the lacrimal gland.

Slit lamp examination of the cornea showed no active involvement. The anterior chamber was free of cells. A subcortical coronary cataract was present, but the fundus was normal.

Examination of the mouth disclosed a gingivitis of moderate severity, but in the general examination no other apparent focus of Vincent's infection was found.

(A description of the left eye was facilitated by showing some pictures.)

Smears from the left eye showed numerous fusiform bacilli, occasional spirilli, gram-positive cocci in pairs and chains and diphtheroids.

Smears from the infection of the gums revealed a similar variety of organisms, with large numbers of fusiform bacilli.

Aerobic cultures from the left eye, taken on rabbit's blood agar, produced Streptococcus viridans, Staphylococcus aureus, Streptococcus haemolyticus and diphtheroids.

The results of dark field examination and special anaerobic cultures were made available by Dr. Thygeson and Dr. Khorazo.

Dark field examination showed a variety of fusiform bacilli, highly motile vibrios and the spirochetes Spirochaeta macrodentium and Spirochaeta microdentium.

Anaerobic culture in ascitic fluid with added gentian violet grew a pure culture of fusiform bacilli. One attempt to grow the spirochetes met with no success.

The treatment has been largely disappointing. Local application of a 3 per cent solution of neoarsphenamine in glycerin for one week supported with two injections of 0.3 Gm. of neoarsphenamine intravenously seemed to aggravate the condition and increase the extent of the pseudomembrane.

Irrigations with a 1 per cent solution of sodium perborate apparently aided in the removal of the pseudomembrane but produced so much chemosis and irritation that its continuance was deemed inadvisable.

The patient is now receiving an ointment containing mercury bichloride (1 : 3,000), as has been suggested by Dr. Macnie. There seems to be some decrease in the amount of discharge and a slight diminution in the number of fusiform bacilli seen in the smear. The focus of infection in the mouth is now being energetically treated by a competent dentist.

Conclusion.—This patient has bilateral conjunctivitis with a clinically similar infection in each eye. While complete bacteriologic study of the right eye is not available, it would seem likely that this eye was lost as a direct complication of fusospirochetal disease, such as is now present in the left eye.

I believe that the apparent involvement of the palpebral lobe of the lacrimal gland is probably secondary to the conjunctivitis. However, as the greatest amount of reaction and discharge appears consistently to be at this point, it would seem possible that a chronic infection of the gland could be a factor in reinfecting the conjunctiva and rendering the local treatments ineffective.

Any suggestions which may lead to a more effective treatment of the patient will be more than welcome.

Plastic Material for Implantation into Orbit After Enucleation.
DR. CONRAD BERENS and DR. SIDNEY ROTHBARD (by invitation).

Although the implantation of gold balls has proved more satisfactory in our experience than fat or other substances, the movements of the prosthesis have been limited at times, and gold balls are expensive. Therefore, experiments with various shapes and sizes of plastic material have been performed.

The implants (made by V. Mueller & Co.) are obtainable in sizes of 12, 14, 15, 16 and 18 mm. The form selected as the most desirable is a quadrilateral, pyramid-shaped piece of plastic material, rounded at one extremity and tapered to a blunt point at the other. The implants may also be obtained in the form of spheres and cones.

The material is easily sterilized by boiling or by placing it in a cold solution, such as alcohol or other antiseptics. Immersion for ten minutes in one of the following solutions is recommended for sterilization: merthiolate (1 : 1,000), hexylresorcinol (1 : 1,000), metaphen (1 : 500) or mercury bichloride (1 : 400).

This plastic material seems to be nonirritating both for animals and for human beings. Implants have not been in place in the human or animal orbits for more than nine months. Therefore, it is too early to draw final conclusions concerning the ultimate value of this material in regard to its nonirritating qualities to human tissues and possible absorption. However, no histologic evidence of harm to the tissues was observable.

It is also too early to state that an implant of quadrilateral pyramidal shape may not have certain disadvantages, for only nine implants have been used after enucleating human eyes. It seems as though such an implant might tend to stretch the muscles more than one that was spherical and that pivoting on the point of the implant should result

in better average movability of the prosthesis. The fact that the material cannot be broken, as glass spheres have been known to be, justifies further trial of such implants and further studies of the shape.

This study, from the department of research of the New York Eye and Ear Infirmary and the department of pathology of Cornell University Medical College, was aided by a grant from the Ophthalmological Foundation, Inc.

DISCUSSION

DR. SIDNEY ROTHBARD: I think I can give an idea of the effect the synthetic plastic material had on the animal body.

Chemically, this plastic is a polymerized methylmethacrylate resin. Specimens that were accurately weighed and measured were placed under aseptic conditions in the subcutaneous tissues, pleural and peritoneal cavities and in the anterior chambers of the eyes of rabbits. The animals were permitted to live for three to nine months. There was no evidence of an inflammatory reaction during the period of observation. The rabbits gained weight, appeared alert, were active at all times and showed no changes in the fundi.

The plastic material which was removed at autopsy did not reveal any change in size or shape during the experimental period. A thick-walled, well vascularized fibrous capsule surrounded each specimen. No cellular reaction was noted in this tissue when it was examined histologically.

Autopsy on the animal organs and tissues gave essentially negative results. The heart, lungs, liver, spleen, kidneys, adrenals and eyes were examined microscopically. No abnormality was noted in any of the organs or tissues.

From these findings it is apparent that the material has no effect on the animal body in any way, and in turn the animal tissue has no effect on the material used during the period of observation.

Autokeratoplasty. DR. WENDELL L. HUGHES.

A method of autokeratoplasty, whereby a rectangle of cornea 4 by 8 mm. is removed and turned end for end, is applicable in cases in which there is a central corneal scar with a clear area of cornea in the periphery. This maneuver places the clear section of the cornea over the pupillary area and the opaque area peripherally.

(Colored motion pictures of this procedure were shown to demonstrate the technic.) In 1 case the procedure was used on an amblyopic eye. In 3 other cases the operation was carried out with good results.

By using the patient's own cornea, there will be less tissue reaction than with a piece of foreign cornea. The procedure is, of course, indicated only in cases in which there is sufficient clear cornea peripherally to provide a useful clear zone centrally.

DISCUSSION

DR. JOHN CUNNINGHAM: Dr. Castroviejo, who was asked to discuss this paper, cannot be here and has asked me to take his place.

The technic of rectangular autotransplantation by rotation of the graft has been tried by Dr. Castroviejo in the past and by myself in the past four months on 15 rabbits. The results thus far obtained suggest

giving up this type of operation unless material from a donor cannot be obtained. We make this suggestion for the following reasons:

1. Unless a preliminary iridectomy is done, incarceration of the iris in the periphery is likely to occur.

2. Healing of such a large graft is often uneven, and a nebulous graft results.

3. Secondary glaucoma resulted too often due to peripheral anterior synychia, since in this operation a fair share of the transplant is directly over iris.

I quote Dr. Castroviejo: "In regard to autotransplants and homotransplants, experimental work on animals' eyes showed that if a corneal flap is dissected free and allowed to heal in the same place, the postoperative course is the same as that which occurs in homokeratoplasty.

"Von Friede in 1936 reported 8 cases of autokeratoplasty. One cornea became partially opaque, and 2 operations were complete failures. The percentage of failures in cases reported by von Friede is too high to justify the performance of this type of operation in such cases when good results may be expected when suitable technic of homokeratoplasty is used."

The use of egg membrane or rubber tissue as possible protective coverings has been worked on experimentally by Dr. Castroviejo during the past three or four years and has been given up because it was found to complicate the operation and did not prevent the formation of abscesses in the cornea of the host or the tendency to interstitial vascularization when sutures are left longer than six days.

The assumption that an autoplastic graft should result in less reaction and that a graft of foreign tissue would naturally be more likely to degenerative changes, as Dr. Hughes has stated, must be subjected to further experimental proof. Thus far our studies do not confirm this assumption. Our completed study will be published at a later date.

A Simple Suture for Retention of Corneal Graft. Dr. Sigmund A. Agatston.

Following Castroviejo's technic of corneal transplantation, the retaining criss-cross suture was found cumbersome. Furthermore, it added to trauma by undue manipulation.

A simple method was devised consisting of two diagonal mattress sutures, across the angles. The components of each suture should be 3 mm. apart. The suture is kept loose until the graft is placed. At the end of the operation sutures are drawn taut in purse-string fashion. The angles are thus successfully held in place.

Observations on the Innervation and Pharmacology of the Ocular Muscles. Dr. Morris Bender (by invitation).

Movements of the intrinsic and extrinsic ocular muscles are accomplished by excitatory and inhibitory neural mechanisms. Chemical agents released within the body influence the activity of these muscles. Epinephrine, the sympathomimetic hormone, acts on those muscles innervated by sympathetic or "adrenergic" nerves—those nerves which liberate epinephrine at their endings, e.g., the pupillodilator fibers of the iris. Acetylcholine, the parasympathomimetic hormone, acts on the striated

ocular muscles or those structures innervated by parasympathic or "cholinergic" nerves—those nerves which secrete acetylcholine at their endings, for instance, the pupilloconstrictor fibers of the iris.

When muscles are denervated postganglionically they become hypersensitive to their respective chemical mediators (law of denervation). Thus the presence of an excess of "adrenergic" hormone is indicated by a dilatation of the pupil in the eye in which the radial fibers of the iris have been denervated by removal of the superior cervical ganglion. The "cholinergic" hormone can be detected by denervated pupilloconstrictor or extrinsic ocular muscles, either of which react with infinitesimal amounts of acetylcholine.

With these denervated structures of cats and monkeys as indicators, sympathetic and parasympathetic agents are found to be released within the body during fright (Bender: *Am. J. Physiol.* **121**:609, 1938; **127**:15, 1939. Bender and Fulton: *J. Neurol. & Psychiat.* **2**:285, 1940) as well as insulin hypoglycemia (Bender and Siegal: *Am. J. Physiol.* **128**:324, 1940) in increased quantities. Under normal conditions, these substances are released at respective nerve endings or from other sources into the general circulation and thus have a direct and continual influence on the innervated muscles, even though there is no visible reaction exhibited by denervated muscles.

Retrobulbar Neuritis: Observations on One Hundred Cases. Dr. Frank D. Carroll.

This article was published in full in the July issue of the Archives, page 44.

David Webster, M.D., *Chairman*

Robert K. Lambert, M.D., *Secretary*

May 20, 1940

Necrosis of the Cornea Due to Vitamin A Deficiency. Dr. H. Krieger Goldberg (by invitation) and Dr. Kaufman Schlinek.

This paper will be published in full, with discussion in a later issue of the Archives.

Traumatic Retinal Angiopathy. Dr. James W. Smith.

G. L., a man aged 46, sustained a chest compression injury, right hemothorax and fracture of the right second, fifth and sixth ribs and fracture of the left frontal bone in an automobile accident. The right optic nerve appeared pale, and retinal hemorrhages and edema were observed by the attending neurologist after the accident. When the patient was first examined ophthalmoscopically ten days later, the fundus was obscured by a diffuse, foglike veil which occupied the posterior two thirds of the vitreous. Ten months elapsed before the

entire fundus was again visible. Sixteen months after the accident vision equaled 4/200, limited to the superior nasal field. The optic nerve appeared atrophic. Changes secondary to hemorrhage were seen about the macular zone and about the inferior nasal vein. A thin, semitransparent vitreous veil was seen in front of the optic nerve.

The left optic nerve was pale, the retina was normal and final vision equaled 15/200. A hemorrhage far back in the orbit with subsequent clot formation probably produced partial atrophy of the optic nerve.

DISCUSSION

DR. RALPH I. LLOYD: I do not think Dr. Smith emphasized sufficiently the importance of compression of the chest in this case. I have been present at 2 autopsies performed on persons after similar automobile accidents, and in both instances there was actual trauma of the lung with hemorrhage from the compression of the chest by pressure of the steering wheel. The resulting venous stasis is really an asphyxia.

Just how an injury to the head produces intense venous congestion of the brain I do not know, but it is this venous stasis that pathologists have described as the particular feature of this type of smothering.

DR. JAMES W. SMITH: Dr. Rosenheck has sent in a few remarks on the neurologist's conception of the ocular changes which are pertinent to the discussion:

"In spite of the thousands of skull injuries that have been observed, the fundi oculi miraculously escape. One may see minor changes as evidence of interference with blood supply, but, generally speaking, nothing very serious occurs. I am not unaware of the fact that other men have reported on series of head injuries with traumatic retinal changes. When they take place and produce Purtscher's phenomenon, other factors are obviously present. What these are is more or less speculative, but it is more than an assumption neurologically that sudden increase of the intracranial ventricular tension plus interference with return flow to the thoracic veins from the head, as occurs in chest compression, plays the deciding role in the etiology of traumatic retinal angiopathy.

One would naturally question this and ask why papilledema is not present in these cases if increased intracranial pressure is a factor. This is explained by stating categorically that this pressure is not sustained long enough to produce choked disk. The blow and its effect are instantaneous and disappear almost momentarily, so that the intracranial pressure returns to normal and does not compromise the intervaginal space of the optic nerve and the central retinal vein within the nerve."

Recurrent Advanced Carcinoma of the Orbit and Nose: Electrocoagulation and Plastic Reconstruction. DR. TIBOR DE CHOLNOKY.

A 53 year old man had a recurrent basal cell carcinoma which destroyed the left eye and involved the frontal sinuses and the left antrum. The originally small lesion at the left inner canthus was inadequately treated with radium.

After electrothermal destruction, plastic reconstruction was done. A prosthesis was used for the eye. There was no recurrence for three years.

This presentation is made to show the effectiveness of electrocoagulation in advanced carcinoma followed by plastic reconstruction.

Streptotrichosis of the Lacrimal Canaliculi. Dr. Alfred Johnston Elliot (by invitation).

In 7 cases of streptotrichosis of the lacrimal canaliculi seen over a two year period at the New York Eye and Ear Infirmary, pus at the inner canthus and tearing were the chief complaints. Either concretions or mucopurulent material was obtained from the canaliculi. Both upper and lower canaliculi were involved in 2 cases, while in 1 case only the upper canaliculus was affected. Treatment included slitting of the canaliculus, dilation and curettage. Compound solution of iodine was instilled into the canaliculus, and saturated potassium iodide, 10 drops three times a day, was given by mouth.

DISCUSSION

Dr. Kaufman Schlivek: The condition described by Dr. Elliot is not uncommon. The only difficulty is that so frequently the diagnosis is overlooked, but it is simple enough to make if one keeps the condition in mind. Cure can be accomplished in almost every case by one treatment. The therapy consists of dilation of the punctum, expression of the secretion or concretion (the fungous mass), removal of the rest of the secretion in the canaliculus by curettage and, finally, application of 3.5 per cent tincture of iodine directly into the canaliculus. This can be done by sharpening a toothpick and wrapping it with cotton, just enough to thoroughly apply the tincture of iodine. This application is followed by the use of a little alcohol. Care should be taken to put plenty of ointment into the conjunctival sac so that the excess of iodine will not irritate the conjunctiva. Invariably one treatment of this kind will result in cure. If one feels that there is a possibility of recurrence, potassium iodide given for a few weeks will ease one's mind, but the condition will not recur.

Dr. Alfred Johnston Elliot: In regard to Dr. Schlivek's comments on treatment with tincture of iodine, my patient who had concretions in the upper canaliculus only was treated for a considerable time by the method outlined, and streptotriches are still present.

Dr. Kaufman Schlivek: Were dilation and curettage done?

Dr. Alfred Johnston Elliot: Yes, those procedures were carried out, and iodine was instilled.

Congenital Ptosis: Corrected By the Machek Operation. Dr. Rudolf Aebli.

An 11 year old girl had congenital ptosis of the left eye. The left levator palpebrae and both elevators of the left eye were almost completely paralyzed. The child had been operated on four times during the past two years. First a Gillet de Grandmont operation was performed, and then a Blaskovics resection of the levator muscle; subsequently resection of the superior rectus muscle was performed, followed by a Motais operation. The child still had marked ptosis. Since the slight elevation that was produced was done by the frontalis muscle,

and it was felt that an operation that would effect more intimate adhesions between the lid and this muscle was indicated. The Machek operation was performed according to the technic and modifications described by Gifford in the October 1932 issue of the ARCHIVES (page 495). This gave a satisfactory result.

Molluscum Contagiosum of the Margin of the Lid as a Cause of Follicular Conjunctivitis. DR. PHILLIPS THYGESON.

Unilateral follicular conjunctivitis of two and one-half months' duration healed rapidly after removal of a molluscum nodule from the margin of the lower lid. The diagnosis of molluscum contagiosum was made by the finding of typical elementary bodies in expressed tumor material stained with victoria blue and by histologic examination of the excised nodule. I have observed 4 other cases of molluscum contagiosum; the associated conjunctivitis was catarrhal in 2 and follicular in 2, severe keratitis with pannus also being present in 1 of the latter 2 cases.

DISCUSSION

DR. KAUFMAN SCHLIVEK: I should like to add one point. It is not necessary that the molluscum be at the margin of the lid. It may be fairly remote, under the brow or under the lower lid. This subject was brought to the attention of the American medical profession originally by Dr. Harold Gifford. He wrote an article some years ago, and his theory was that by rubbing the eye the infection was carried into the conjunctival sac.

DR. G. BONACCOLTO: In 1927 I had the opportunity of seeing a case of molluscum contagiosum while in training with Professor Bardelli.

The patient had been treated elsewhere for trachoma, and in the clinic there were few who could make a differential diagnosis between trachoma and any other type of follicular conjunctivitis. The condition was diagnosed by Professor Bardelli as molluscum contagiosum, since he had had occasion to see other similar cases previously, reports of which he had published.

In this particular case the molluscum nodule was not at the margin of the lid, as in the case presented by Dr. Thygeson, but about 1 cm. below its margin, and in spite of its umbilicated appearance, it was taken for a verruca by most of those who saw it, and the conjunctivitis was mistaken for trachoma.

After the expression of the molluscum, the conjunctivitis cleared up.

Traumatic Cataract Affecting the Anterior Layer of Congenital Nucleus. DR. G. BONACCOLTO.

A case of traumatic cataract affecting the anterior layer of congenital nucleus was presented.

Sarcoma of the Choroid: Report of Cases. DR. WALTER HIPP.

CASE 1.—Miss B. F., a 30 year old white woman, came to my office on March 23, 1940 complaining that vision in the left eye was failing.

External examination gave negative results. Vision without correction was 20/200 in the right eye and 20/40 in the left. With glasses, it

was 20/40 in the right eye and 20/30 in the left. Reduced vision in the right eye was accounted for by the fact that the patient has a high astigmatic correction in the left eye; however, a + 50 cylinder was accepted, and the best vision was 20/30.

Ophthalmoscopic examination showed no pathologic process in the right eye, but in the left eye there was a grayish black mass protruding into the vitreous slightly above to the temporal side of the macula.

A diagnosis made was a melanosarcoma of the choroid. This diagnosis was corroborated by at least twenty-five other physicians (ophthalmologists).

The eye was removed, and so far the pathologic specimen has not been reported on.

CASE 2.—Mr. W. D. came to see me in June 1939 complaining of the fact that he saw horizontal lines at an angle with the right eye. His refraction was checked, and no change was necessary in his glasses, which gave him a vision of 20/15 in both eyes.

Ophthalmoscopic examination showed a slight grayish mass close to the macular area and protruding into the vitreous. A diagnosis of sarcoma of the choroid was made.

Probing of the Lacrimal Canal with Negative Pole. DR. WALTER HIPP.

Miss A. A. came to my office in July 1939 complaining of tearing of the right eye. She stated that her duct had been probed and irrigated many times but always closed again. The eye was anesthetized with a 0.5 per cent solution of pontocaine; the punctum was dilated with a punctum dilator, and a no. 2 probe, to which was attached the negative pole of galvanic current, was inserted into the punctum, through the duct and down into the nose. The duct was then irrigated with physiologic solution of sodium chloride, which flowed freely into the nasal cavity.

This procedure has been repeated three times, and for the past year the patient has had no further trouble.

DISCUSSION

DR. A. L. KORNZWEIG: Has Dr. Hipp ever treated any patients with stenosis of the nasolacrymal duct in this way with good results?

DR. BENJAMIN FRIEDMAN: I should like to ask Dr. Hipp whether by the negative pole he means the indifferent electrode of the diathermy machine or the negative galvanic current.

DR. WALTER HIPP: I mean the galvanic current.

In reply to Dr. Kornzweig, I have 1 case in which this form of treatment has been used to a certain extent, but the result does not seem to be altogether good. The duct was probed time and again, and while there was not perfect closure every time it was more difficult to get the probe through, but now it is easier and simpler. The condition has not entirely cleared up.

Birth Injury of the Cornea with "Glass Membrane" in the Anterior Chamber. DR. CHARLES A. PERERA.

This paper will be published in full, with discussion in a later issue of the ARCHIVES.

Book Reviews

Traité d'ophtalmologie. Published under the auspices of the Société française d'ophtalmologie, by P. Bailliart, C. Coutela, E. Redslob, E. Velter and R. Onfray, general secretary. Eight volumes, pp. 8,058, with 176 plates in color. Price, 2,800 francs. Single volume, 350 or 400 francs. Paris: Masson & Cie, 1939.

Volume 7 contains 938 pages, 414 figures and 31 plates in color and has the following contents:

The section entitled "Ocular Neurology" is continued and the titles of the chapters are: "Oculo-Motor and Pupillary Disturbances," by E. Velter; "Strabismus," by René Onfray; "Nystagmus," by Henri Coppez; "Ocular Disturbances After Lesions of the Fifth Nerve," by L. Cerise and Thurel; "Ocular Symptomatology in Diseases of the Central Nervous System," by E. Velter; "Neurosurgical Diagnosis of Lesions in the Chiasmal and Sellar Regions," by Clovis Vincent and Pierre Puech, and "Diagnosis and Treatment of Intracranial Hypertension and of Tumors of the Brain—Ventriculography," by T. de Martel and J. Guillaume.

The chapter on strabismus (pages 31 to 137), by Onfray, gives an exhaustive and up to date study. It is interesting to learn the views of the French school on this important subject, a subject which received such an impetus from the early investigations by Landolt.

The diseases of the fifth nerve are comprehensively treated on pages 178 to 271 and include neuralgia, neuroparalytic keratitis, zoster and herpes.

The chapter on neurosurgical diagnosis of diseases of the chiasmal and sellar regions, by the celebrated French neurosurgeon Clovis Vincent, is excellent.

The second half of the volume is devoted to "Ocular Surgery," and the subject is divided into the following chapters: "Surgical Generalities," by R. de Saint-Martin; "Operations on the Eyelids and Conjunctiva," by C. Duverger and E. Velter; "Operations on the Lacrimal Apparatus," by P. Merigot de Treigny; "Operations on the Cornea, Sclerotic and Iris," by C. Duverger and E. Velter; "Extracapsular Extraction of Cataract," by A. van Lint; "Intracapsular Extraction of Cataract," by R. de Saint-Martin; "Operative Treatment of Dislocation of the Lens," by R. de Saint-Martin; "Operations for Detachment of the Retina," by Prosper Veil and M.-A. Dollfus; "Operations for Glaucoma," by C. Duverger and E. Velter; "Treatment of Strabismus: Surgical Technic," by R. de Saint-Martin and René Onfray; "Enucleation: Resection of the Anterior Segment of the Eyeball; Intrascleral and Intraorbital Implantation," by P. Carlotti; "Operations on the Orbit," by F. Terrien; "Treatment of Traumatisms and of Foreign Bodies in the Eye and in the Orbit," by C. Duverger and E. Velter, and "Prothesis," by J. Bruneau and J. Charpentier.

De Saint-Martin introduces the subject of ocular operations by an excellent chapter on surgical generalities as applied to the eye. Then the operations are taken up according to anatomic subdivisions by ophthalmic surgeons whose names are well known in ophthalmic literature. Thus, Duverger and Velter handle operations on the eyelids and conjunctiva, cornea, sclera and iris and for glaucoma and intraocular foreign bodies. To van Lint, the eminent Belgian surgeon, whose work on akinesia is so well known, has been entrusted the extracapsular extraction of cataract, while de Saint-Martin, who has recently covered the subject of cataract operation in a monograph of the French Ophthalmological Society, describes the intracapsular extraction. Detachment of the retina, that new chapter in ophthalmic operations, is from the pen of Veil and Dollfus, of the Hotel Dieu in Paris. Finally, operations for strabismus are described by de Saint-Martin.

Volume 8 contains 1,000 pages, 164 illustrations and 2 plates in color, and takes up the subjects of therapeutics and hygiene. A section on medical therapeutics is divided into the following headings: "The Technic of Local Medication," by J. Plicque; "Mendicamentous Therapeutics," by R. Margerin; "Biologic Therapeutics," by J. Plicque; "Physical Agents, Excluding the Roentgen Rays," by P. Reiss; "Radiotherapy," by A. Gunsett; "General Indications and Therapeutic Synthesis," by J. Plicque; "Eyeglasses," by H. Liégard, and "Contact Glasses," by Emile Haas.

The subject of hygiene and industrial injuries occupies the second part of this volume. The chapters are: "Individual Hygiene" and "Hygiene in the Schools," by P. Pesme; "General Hygiene," by R. Bonnardel; "Contusions," "Injuries and Foreign Bodies" and "Burns," by J. Chappé; "Injuries from Dust and Vapors and Gas, and Industrial Poisoning," "Infections and Parasitic Industrial Diseases and Electrical Injuries," "Ocular Fatigue and Care" and "Public Hygiene (Reportable Diseases)," by C. Coutella; "Visual Requirements," by A. Cantonnet, and "Blindness," by A. Monthus.

The volume concludes with the following chapters: "Medicolegal Ophthalmology," by C. Coutella; "Tropical Ophthalmology" and "Parasitic Diseases of the Eye," by P. Toulant; "Comparative Ophthalmology," by Robin and Brion, and "Deontology and Ophthalmology," by F. Cosse and Gaillaud.

An alphabetical table of contents of the eight volumes (pages 880 to 1002) conclude this volume.

The reader will find in this handbook a complete survey of the many parts of ophthalmology, arranged in a logical manner, written in a clear, concise style, interesting, never boresome and excellently illustrated.

The publication committee of the French Ophthalmological Society deserve great credit not only for the admirable selection of the contributing authors but especially for the promptness of the appearance of the publication. The handbook is a monument to French ophthalmology, and the publishers, Masson & Cie, are to be complimented on the beauty of the book making and for the reasonableness of the price.

ARNOLD KNAPP.

Lehrbuch der Augenheilkunde by Dr. Ernst Fuchs. Edition XVI, enlarged and improved. By Dr. Adalbert Fuchs, Professor of Ophthalmology, University of Vienna. Pp. 904, with 362 illustrations, many in colors and 5 colored plates. Price, 27 marks. Vienna: F. Deuticke, 1939, with a replica of Wüllner's superb bas-relief of E. Fuchs.

Fuchs's textbook, which has served so many ophthalmologists as a book of instruction and later as a constant book of reference to which one returns in any emergency for help and advice, has been appearing regularly since 1889. The thirteenth to fifteenth editions were revised by Professor Salzmann, of Graz, who in addition to other changes, added original drawings of the fundus in color. When a new edition, the sixteenth, became necessary, fortunately A. Fuchs, the talented son of the author, was prevailed on to undertake the revision. He has succeeded in broadening the scope of the book by enlarging the part dealing with conditions of the fundus and their relation to general diseases. The chapters on tumors, neurology and muscles have been revised and brought up to date. Importance is given to inherited diseases, and the nonoperative and medical treatment of ocular diseases has been given special attention. The new additions are on injuries due to war gases and on the important subject of detachment of the retina.

The book has been particularly enriched by many excellent drawings which the author has taken from his own atlas on the pathologic anatomy of the eye.

Professor Fuchs has succeeded admirably in adding to the rich treasures which had made his father's book famous and by bringing the contents up to date. Fuchs's textbook should continue to occupy the foremost position in the world's books on ophthalmology.

ARNOLD KNAPP.

Indian Journal of Ophthalmology. Managing Editor, Vaman D. Sathaye, Bombay. Editorial Committee: C. B. Dhurandhar, Bombay; S. V. Oak, Bombay; D. G. Patwardhan, Poona; P. V. Pendse, Baroda, and Vaman D. Sathaye, Bombay. Annual subscription price, 6 shillings. First number, January 1940; published quarterly. Editorial office, 502 Narayen Peth, Poona City, India.

In the foreword it is stated that a great country like India, which gave birth to ophthalmic surgery, should have a journal of ophthalmology of its own, and as the problem of blindness in India is an acute one, the editors have been induced to make this undertaking.

The present number contains the following four original articles: "Extraction of Subluxated Lens, Made Nonviolent by Lever Action Intracapsular Operation," by Kartik Chunder Dutt; "Foreign Bodies of the Eye: Some Observations," by Shafi Mehkri; "Phlyctenular Keratoconjunctivitis: Its Etiology and Treatment," by Sridhar V. Oak, and "Correcting Spastic Entropion of the Upper Lid Following Total Tarsectomy, by Transplanting a Shaving from Concha Auriculae," by H. L. Anand.

In addition, there are clinical notes and abstracts.

ARNOLD KNAPP.

Directory of the Medical Specialists Certified by American Boards, 1939. Price, $5.00. Pp. 1,573. New York: Columbia University Press, 1940.

This is an official directory of 14,000 specialists who have been certified by the American Boards and replaces the individual lists of the twelve examining boards. The new directory brings much additional information, such as hospital appointments, teaching positions and society memberships. In the first section the purposes of the Advisory Board for Medical Specialties are described, and separate sections are devoted to the special boards, with the list of officers and the examination requirements of each given in detail. Finally, an alphabetical list of diplomates is given.

This publication is priced at cost; as it is in line with improving graduate medical education, it merits approval and deserves every one's cooperation.

It is planned to issue a revised edition every two years. The directing editor is Dr. Paul Titus; the associate editor, Dr. J. Stewart Rodman, and the advisory editors, Dr. Paul M. Wood, Dr. C. Guy Lane, Dr. William S. Middleton, Dr. John Green, Dr. Fremont C. Chandler, Dr. W. P. Wherry, Dr. F. W. Hartman, Dr. C. Anderson Aldrich, Dr. Walter Freeman, Dr. B. R. Kirklin and Dr. Gilbert J. Thomas.

The directory should have a wide distribution, as it will be found most useful.

ARNOLD KNAPP.

Bulletin of the Ophthalmological Society of Egypt. Volume XXXII. Session 36, 1936. Pp. 312. Cairo: Imprimerie Misr, S. A. E., 1940.

The thirty-sixth annual congress of the Ophthalmological Society of Egypt was held on March 24 to 25, 1939, at the Memorial Ophthalmic Laboratory in Giza. The president, Dr. Mohammad Mahfouz Bey, opened the congress, and a number of interesting communications were presented, which included one on ocular lesions due to infection with Onchocerca in Egypt, by Dr. M. Khalil Bey, and a symposium on non-suppurative keratitis.

In addition, Dr. Rowland P. Wilson gave a preliminary report on the treatment of trachoma with sulfonamide compounds; Dr. M. Khalil described keratoplasty; Dr. Elia Nashed spoke on quinine amblyopia, and Dr. P. Sander reported on a visit of Dr. Mathra Dass, of Lahore, India, to the Indian hospitals and the Gojra Civil Hospital, of which Dr. Harshajian Singh is director.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.
Place: Heidelberg. Time: Aug. 5-7, 1940.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.
Place: Perth, Western Australia. Time: Sept. 2 and 7, 1940.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIEDADE DE OPHTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Francisco Ferreira, Pitangueiras 15, Brotas, S. Salvador, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 IIII tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Albert C. Snell, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary: Dr. Derrick Vail, 441 Vine St., Cincinnati.
Place: Cleveland.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Cleveland. Time: Oct. 6-11, 1940.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. F. P. Calhoun, 478 Peachtree St. N. E., Atlanta, Ga.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. John Evans, 23 Schermerhorn St., Brooklyn.
Secretary-Treasurer: Dr. C. S. O'Brien, University Hospital, Iowa City.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St.; Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President-Emeritus: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.
Executive Director: Mrs. Eleanor Brown Merrill, 50 W. 50th St., New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Charles W. Barkhorn, 223 Roseville Ave., Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Nadeau, 122 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Green Bay. Time: October 1940.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. William D. Rowland, 84 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles, Calif.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Clarence W. Shannon, 4th and Pike Bldg., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1215–4th Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. E. Rideout, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. J. J. Potter, 303 N. Main St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. Kessler, 311 Center Ave., Bay City, Mich.
Secretary-Treasurer: Dr. J. H. Curts, 330 S. Washington Ave., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Davis, 1615 Howard St., Omaha.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux Falls, S. D.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. Dake Biddle, 123 S. Stone Ave., Tucson, Ariz.
Secretary: Dr. A. E. Cruthirds, 15 E. Monroe St., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. J. K. Heckert, 103 N. Washington St., Lansing.
Secretary-Treasurer: Dr. W. D. Irwin, 710 Hanselman Bldg., Kalamazoo.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Hugh B. Barclay, 111 S. Main St., Greensburg.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. Raymond C. Cook, 701 Main St., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. William M. Bane, 1612 Tremont Pl., Denver.
Secretary: Dr. Harry Shankel, Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. W. E. McClellan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. E. N. Maner, 247 Bull St., Savannah.
Secretary-Treasurer: Dr. C. K. McLaughlin, 567 Walnut St., Macon.
Place: Macon. Time: May 13-17, 1941.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. K. Von Lackum, 117–3d St. S.E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Cedar Rapids. Time: November 1940.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Henry N. Blum, 912 American Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. O. B. McGillicuddy, 124 W. Allegan St., Lansing.
Secretary: Dr. R. G. Laird, 116 Fulton St., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Erling Wilhelm Hansen, 78 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Edgar P. Cardwell, 47 Central Ave., Newark.
Secretary: Dr. Arthur E. Sherman, 243 S. Harrison St., East Orange.
Place: Atlantic City. Time: June 3-5, 1941.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Searle B. Marlow, 109 S. Warren St., Syracuse.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. A. G. Woodward, 100 S. James St., Goldsboro.
Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Secretary-Treasurer: Dr. R. S. Fixott, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. W. Carpenter, 200 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia. Time: Nov. 5, 1940.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. J. W. Ward, 2607½ Lee St., Greenville.
Secretary: Dr. Dan Brannin, 929 Medical Arts Bldg., Dallas.
Place: Fort Worth. Time: Dec. 13-14, 1940.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. A. E. Callaghan, Boston Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Rowland H. Merrill, 1010 First National Bank Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. George G. Hawkins, Newport News.
Secretary-Treasurer: Dr. Guy Fisher, 3 E. Beverley St., Staunton.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George E. Black, 7 W. Bowery St., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Stacy C. Howell, 144 Ponce de Leon Ave. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther Wilson, Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Maurice Wieselthier, 1322 Union St., Brooklyn.
Secretary-Treasurer: Dr. Harold F. Schilback, 142 Joralemon St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. James G. Fowler, 412 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Richard Gamble, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmologic Club

Chairman: Dr. Albert D. Ruedemann, Cleveland Clinic, Cleveland.
Secretary: Dr. B. J. Wolpaw, 2323 Prospect Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Francis H. Adler, 313 S. 17th St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. D. Postle, 240 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Hugh C. Thompson, 289 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. F. K. Stroud, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. Arthur Padilla, 414 Medical Professional Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Dan Brannin, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. L. E. Darrough, 4105 Live Oak St., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. A. E. Jackson, 602 W. 10th St., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. Charles Dickson, 1617 Medical Arts Bldg., Houston, Texas.
Secretary: Dr. William J. Snow, 708 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. M. Masters, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Homer Beal, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Desmond Curran, Federal Reserve Bank Bldg., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Paul Southgate, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Kirt G. Parks, 605 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. Colby Hall, 1136 W. 6th St., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. E. J. Cummings, 1835 I St. N. W., Washington.
Secretary: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Secretary-Treasurer: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Secretary: Dr. Guy Maness, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. Leonard C. Whiting, 121 Whitney Ave., New Haven, Conn.
Secretary: Dr. Frederick A. Wies, 255 Bradley St., New Haven, Conn.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Algernon Reese, 73 E. 71st St., New York.
Secretary: Dr. Brittain Payne, 35 E. 70th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President Dr. Morris Davidson, 80 Centre St., New York.
Secretary: Dr. Benjamin Esterman, 515 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Leatherock, Cushing, Okla.
Secretary: Dr. Harry C. Ford, 1014 Medical Arts Bldg., Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. Howard Heine, 635 N. Main St., Fremont, Neb.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. L. Markowitz, 16 Church St., Paterson, N. J.
Secretary-Treasurer: Dr. A. John Reinhorn, 302 Broadway, Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward A. Weisser, 119–5th Ave., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. F. Bryce, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

St. Louis Ophthalmic Society

President: Dr. J. F. Hardesty, Missouri Theatre Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Dan Russell, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. P. G. Bowen, 315 Camden St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Matthew Hosmer, 384 Post St., San Francisco.
Secretary: Dr. Fred Boyle, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Louis A. Parsell, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Robert L. Pohl, 407 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. F. R. Webster, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. John R. Myers, State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. S. H. Patterson, 1251–15th St., Toledo, Ohio.
Secretary: Dr. J. E. Minns, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Frank Costenbader, 1726 I St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. L. Conner Moss, 1710 Rhode Island Ave. N.W., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 7:30 p. m., first Monday in November, January, March and April.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.